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VETERINARY MEDICINE

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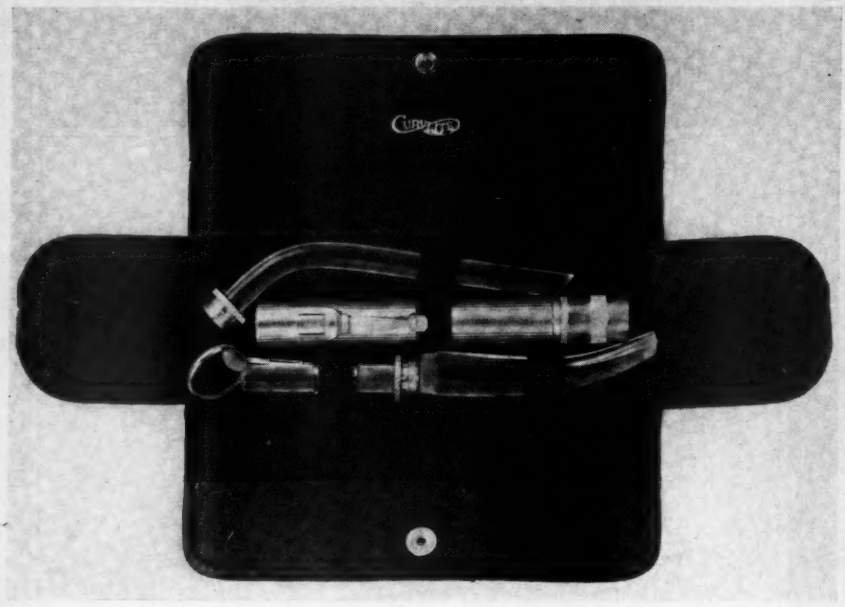

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D. M. CAMPBELL, *Editor*
7632 S. Crandon Ave., Chicago, Ill.
Telephone South Shore 7160

H. J. MOSTYN, *Eastern Representative*
1265 Broadway, New York, N. Y.
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Liver is particularly rich in vitamin B_1 (thiamin) and vitamin G (riboflavin) and is the richest of all tissues in Vitamin A.

* * * *

Liver and lamb's kidneys are the most efficient of all foods in providing the substance necessary to overcome anemia.—*Fishbein.*

* * * *

Nobel Prize to Professor Domagk

The Nobel Prize for Medicine was recently awarded to Prof. Gerhard Domagk, whose name is indelibly associated with the drug sulfanilamide and its derivatives. Domagk was born in Lagow, Germany, in 1895. His principal early work was in the field of pathology. After teaching at Griefswald and Muenster universities, he became director in 1927 of the Institute of Experimental Pathology of the I. G. Dye Works in Elberfeld. Late in 1932 he first demonstrated the curative effects of prontosil (now neoprontosil) in the streptococcic infections of mice. After many careful observations, the details of the new discovery were finally published by Domagk in medical and scientific journals early in 1935. He had earlier received the Emil Fischer Medal, highest award of the German Chemical Society, in 1937 and the Cameron Prize of the University of Edinburgh in 1939.—*Jnl. A. M. A.*, 113:19.

A research project carried out by L. F. Badger of the U. S. Public Health Service led him to the conclusion that as a result of calcium deficiency rats are incapable of utilizing vitamin B_1 in the diet and promptly suffer from vitamin B_1 deficiency also. Such rats are highly susceptible to leprosy

* * * *

Marsh and Wilkins of the Montana Experiment Station investigated a pyogenic facial infection in a band of some 2,000 ewes that had been fed hay during the winter. Of these some 60 died as an indirect result of the disease. The authors concluded that the malady was probably identical to a disease described by Christiansen and others in Europe in 1937, and probably caused by *Actinobacillus lignieresi.*

* * * *

By reason of a recent administrative ruling, medical and veterinary army reserve officers assigned to active duty with the CCC are now employed on a civilian status. Under the new arrangement physicians receive a P-3 rating with minimum compensation of $3,200 a year and veterinarians a P-2 rating with pay initially at $2,600 a year. While on a military status, physicians and veterinarians received the same pay—initially approximately $3,900 for a captain with dependents—with increases for longevity.

An old proverb says, "Tell me what you eat and I'll tell you what you are"—but Hitler, Mussolini and Stalin are vegetarians.

* * * *

Newsom reported the examination of a series of 100 liver abscesses from as many different cattle slaughtered at Denver packing houses. The findings led the author to conclude that most liver abscesses observed in slaughtered cattle are due to the invasion of *Actinomyces necrophorus*, although in an occasional case there may also be *Corynebacterium pyogenes* and a white coccus.

* * * *

New Yearbook of Agriculture Discusses Nutrition Problem

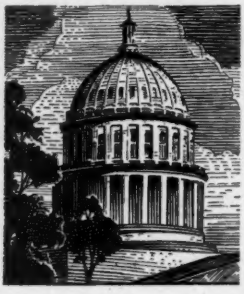
Forty years ago scientists thought the problem of nutrition was fairly simple—solve the needs for proteins, fats, carbohydrates, and a few minerals, analyze foods for these substances, and then so blend the foods as to furnish an adequate supply of each. Now, the scientists know that such knowledge, while fundamental, is a mere introduction to an intricate pattern of known and unknown substances in even the simplest foods. The 1939 yearbook of the U. S. Department of Agriculture, "Food and Life," brings up to date the thoughts on nutrition. First copies of the book are off the press this week.

* * * *

In order to help groups of farmers who desire to form breeding associations where artificial insemination is to be used, a model constitution and a set of by-laws together with various suggestions and cautions have been drawn up jointly by the Farm Security Administration, the Bureaus of Animal Industry and Dairy Industry, and the Extension Service of the Department of Agriculture. For practical application of the new method of breeding the Department recommends that it be tried out only in areas that have a dense livstock population. Experience indicates that it will be practical for livestock breeders within a radius of 10 to 15 miles to form a cooperative breeding association.

Committee on Local Arrangements 1940 A. V. M. A.

The following have been named as members of the Committee on Local Arrangements for the 77th annual convention of the A. V. M. A., to be held in Washington, D. C., August 26-30, 1940: J. R. Mohler, Chairman; A. Eichhorn, Associate Chairman; Hon. G. W. Gillie, Publicity Chairman; H. M. O'Rear, Entertainment Chairman; Mrs. Clifton D. Lowe, Ladies Entertainment Chairman; H. W. Schoening, Educational Exhibit Chairman; Col. J. F. Crosby, Technical Exhibit Chairman; Col. R. A. Kelser, Banquet Chairman; A. E. Wight, Alumni Dinners Chairman; W. H. Wright, President's Reception Chairman; J. P. Turner, Large Animal Clinic Chairman; M. Weadon, Small Animal Clinic Chairman; W. J. Hall, Poultry and Sheep Clinic Chairman.



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Goat's milk, which is popular in Europe, is used in only a few sections of the United States. Three dairies, with approximately 150 goats, supply New York City's demand—250 quarts daily.

* * * *

Goats in the United States

Goats and dogs are more widely distributed than any of the other domesticated animals. There are about 5,000,000 common dairy goats in the United States, with about 500,000 improved goats, and 70,000 good dairy goats. Common goats have a monetary value under $5.00. Improved goats average $20.00, and good goats range up in price from this value. Collectively the dairy goats in the United States are worth about $20,000,000.—*J. C. Marquardt*, in the *Goat World*, 24:11.

The red Leghorn, the first all-red, plumaged fowl, originated in Ohio.

* * * *

B. A. Beach of Wisconsin found that six of 21 cows artificially infected with Bang's disease, recovered as shown by the fact that the blood titers returned to normal. Eighteen of 19 such cows proved resistant to an artificial Brucella infection that caused abortion in 36 of 47 controls. One cow that had passed through a natural outbreak of Bang's disease and recovered was included in the 18. She behaved in exactly the same manner as the others, as far as could be determined.

* * * *

Trichina Infection in Hogs Down 80% in 35 Years

Only about one-fifth as many hogs are infected with trichinae today as were infected some 35 years ago, according to Dr. Benjamin Schwartz, chief of the Zoological Division of the federal Bureau of Animal Industry; based upon a study of the diaphragms of more than 25,000 hogs from the most important swine-breeding areas of the country.

On the basis of the study, about 1.5% of all hogs are infected today as compared to about 7.5% infection in the period 1898 to 1906 when the Bureau examined pork samples from more than eight million hogs.

About 13,000 of all hogs studied were grain fed; about 10,500 had had ordinary garbage as their main food. The rest of the 25,000 hogs had been fed cooked garbage and since they had virtually no trichina infection were not included in the findings.

For the grain-fed hogs the number of diaphragms with trichinae infection was less than 1%, but for the garbage-fed hogs the figure was 5.7%. But because less than 10% of all hogs are garbage fed, infection averages only about 1.5% of all hogs. The majority of the positive hogs had exceedingly light infections.

"In the light of this low infection," says Doctor Schwartz, "it is to be expected that cases of clinical trichinosis in humans are infrequent." The official caution, he says is: "Be sure that all pork is cooked thoroughly."

There is a constantly increasing trend toward pasteurization of milk throughout the United States. At present more than 80% of the fluid milk supply of cities having a population above 75,000 is pasteurized. As the relative size of the city increases, the percentage of the milk that is pasteurized increases; very little raw milk being sold in the large cities. In the smaller towns and rural districts, however, only a small proportion of the milk is pasteurized.—*C. J. Babcock.*

* * * *

Number of Horses Continues To Decline

A further decrease in the number of horses on farms was indicated today by the Bureau of Agricultural Economics of the U. S. Department of Agriculture in its annual outlook report for 1940. Increase in tractor competition, declining prices for horses, inroads of disease in some sections, and a decreased colt crop in 1938 are the bases for the forecast.

The length of time this trend will continue depends largely upon the trend in colt production during the next few years. This trend will be determined by a number of factors—such as the price of work animals relative to prices of other live stock and to tractor prices and prices of feed grains and hay—which will influence farmers' decisions as to the relative returns from colt raising compared with other live stock production.

The number of work stock on farms has decreased steadily since 1913, standing at 10,800,000 horses and 4,382,000 mules on January 1, 1939. Increases in colt production from 1932 through 1937 slowed down the rate of decrease in horse and mule numbers. Colt production decreased in 1938, however, and is now well below the number needed to replace old work stock.

Turning to the foreign situation, the Bureau stated that there is a possibility that the war in Europe may increase exports of horses and mules, but greater use of mechanical units in warfare may make the demand for horses and mules smaller than during the World War.

Milk Production Is Big Business

Of the milk produced in this country, 41% is sold as fluid milk; 41% goes into the production of butter; 8% into cheese; 5% is evaporated; and the remaining 5% is used in the manufacture of ice cream and other milk products. For fluid milk the consumer pays an average price of 12½ cents a quart; the producer receives about five cents a quart, the distribution of fluid milk from dairy to doorstep being one of the most inefficient industries in the country—probably, next to the railroads, the most inefficient large industry. For the milk used in a pound of butter the producer receives an average of 21.4 cents; the consumer pays 34.7 cents for the butter. For the milk used in a pound of cheese, the farmer receives 12.2 cents and the consumer pays 26.5 cents; for the milk in a pound of evaporated milk, the producer receives 2.9 cents and the consumer pays 7.9 cents. For milk used in ice cream and candies the producer receives the lowest price and the consumer pays the highest.

Fifty billion quarts of milk are produced annually in the United States. Of the total production 38,500,000,000 quarts are sold and 11½ billion used on the farm. In 1938 milk and milk products sold for 3½ billion dollars. The total production of the automobile industry sold in the same year for two billion dollars. The total value of the products of the meat packing industry for the same year was two billion 800 million dollars.

U. S. Poster on Bang's Disease

To aid the Federal-State program against Bang's disease, the U. S. Department of Agriculture has just issued a poster on this subject. Besides portraying the disease in pictorial manner, the new poster contains a brief text to inform cattle owners and the public regarding its cause, sources of infection, symptoms, prevention, and eradication. The poster, printed on light cardboard, measures 16 by 20 inches. Copies may be obtained free, as long as the supply lasts, by applying to the Bureau of Animal Industry.

Farm Security Administration officials are encouraging artificial breeding in a number of associations organized for FSA borrowers. About 15 organized groups are now using artificial insemination, but as choice sires are obtained and other obstacles are erased more of the breeding associations are expected to adopt the practice.

All Herds of Cattle in U. S. Tested For Tuberculosis

Every herd of cattle in the United States has now been tested for tuberculosis at least once. The last herd to receive its initial test was in Stanislaus County, California. The first herd of cattle to be officially accredited in the cooperative federal-state campaign against the disease was the U. S. Soldiers' Home herd in Washington, D. C.

In recent months the principal zone of activity against bovine tuberculosis has been in a group of six counties in central California. All other counties in the state and in the United States have already qualified as "modified accredited areas." Since July 1, 1939, a total of 472,599 cattle in California have been tested, of which 8,101 were classed as tuberculous.

During the progress of the nation-wide campaign a total of more than 220,000,000 tuberculin tests have been applied. This number, greatly exceeding the number of cattle in the United States at any time, represents numerous retests and, of course, changes in the herds resulting from births, deaths, and sales of animals for various purposes. During the steady reduction of the disease through systematic testing and removal of reactors, a total of approximately 3,750,000 cattle have been sent to slaughter.

The tuberculosis eradication campaign in the United States has been watched with interest by veterinary and live stock officials in the principal countries of the world, since it is the largest undertaking of its kind in the annals of agriculture and the veterinary profession. The continued activity will consist in periodic retesting in order to detect promptly any cases of the disease, thus providing a safeguard against any material reinfection.

Thirty-Five Years After

In 1904 a small group of persons who were not inclined to run away from a hard job determined to do something about tuberculosis even though there was no known cure

or preventive vaccine for the disease. Only a few health departments were doing anything at all to combat tuberculosis. It was pretty generally accepted that if you had "consumption" you died, and that was all there was to it.

But this little group glimpsed in the distant future the goal that has brought them along the road of sound progress ever since. With eyes and hearts set on the objectives of the dim years ahead, they overcame the inertia of centuries and organized the National Tuberculosis Association.

In 35 years this association has gone far along the road that it could barely discern at the beginning. Today there are 200,000 persons alive in the United States who last year would have died of tuberculosis if that year had been 1904. In 1904, 201 out of every 100,000 persons died of tuberculosis, while in 1938, only 49 out of each 100,000 died of this disease.

You are now urged to buy Christmas Seals to help finance the work of this Association and its nearly 2,500 affiliated organizations in all parts of the country. Organized tuberculosis work considers itself as standing on the threshold at 35 and getting fresh inspiration from the future, as well as pardonable satisfaction from the past.

* * * *

Export of Horses Begins

Contracts have been executed by France for the purchase of 6,000 animals, two-thirds to be riding horses and one-third light and heavy artillery.

They will be bought at only three horse markets: at the Union Stock Yards, Chicago; at the National Stock Yards, Illinois, opposite St. Louis; and at the Kansas City Stock Yards, Kansas City, Missouri.

Riding horses wanted are to be from 15.1 to 16 hands, weighing from 1050 to 1100 pounds; artillery horses are to be from 14.3 to 15.3 hands, weighing from 1150 to 1400 pounds. All are to be from four to eight years old, well broken and better in quality than those accepted in the last war.

Prices paid for these horses will be at present market levels. England has not made any move toward purchases.

If the war is long continued, many more horses may be required. Farm work in France is done principally with horses and mules and many American horses will be needed for crop planting in France in the spring of 1940, if the war holds in service the thousands of animals already drawn from French agriculture.

France had in 1937 a total of 2,742,070 horses; and in 1938 Great Britain, with northern Ireland, had 1,001,526. It is safe to say that all available animals are in the military service, because of the necessity of conserving gasoline for front line fighting planes and tanks.—*Wayne Dinsmore.*

* * * *

Autopsies conducted by Herbert Fox, M.D., on animals dying in the Zoölogical Gardens at Philadelphia during a period of more than 30 years, revealed far more acute endocarditis, particularly of the valves, in the American opossum than in any other species.

43rd Annual Meeting of the U. S. L. S. S. A.

Dr. John R. Mohler, Chief of the Bureau of Animal Industry, U. S. Department of Agriculture, and M. Etienne Létard, professor of Zootechnics at the National Veterinary School at Alfort, France, and head of the French mission to America for the purchase of horses. Prof. Létard, expects to purchase 40,000 animals for the French government. He was introduced at the opening session of the U. S. L. S. S. A. by Doctor Mohler and addressed the members present at some length, complimenting American veterinarians highly on their achievements in tuberculosis eradication, foot-and-mouth disease eradication, and in the control of other diseases.

THE 43rd annual meeting of the United State Live Stock Sanitary Association, held in Chicago, December 6, 7, 8, was a success in point of attendance, quality of the program, and interest. The attendance was somewhat greater than in recent years, the program more extensive, and the attitude of the organization more aggressive. The usual attention was given to tuberculosis eradication, Bang's disease control, rabies, and other measures of predominant interest to live stock regulatory officers.

Dean H. P. Rusk of the College of Agriculture, University of Illinois, made the address of welcome, expressing much appreciation for the service that veterinary medicine has rendered to agriculture in the control of animal disease.

As the bovine tuberculosis eradication project nears completion, interest wanes. The Bang's disease session indicated greater differences of opinion than were shown a year ago, which were considerable even then. The rabies question received extended consideration, the committee on rabies insisting that all measures for suppressing this disease must be used to attain success. They classified these measures under seven groups—six comprising restraint for the dog and the seventh vaccination—and pointed out that all seven are required, and that while restraint measures have been successful in a number of European countries, there is little prospect for their success in this country without the added factor of vaccination. The attitude of the general public, the size of the countries, the density of population, and other conditions which have made it possible to eradicate rabies from a number of European countries without vaccination—England, Norway, Sweden, Denmark, prewar Germany—do not obtain in this country.

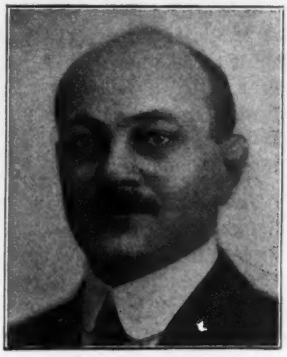
Resolutions and committee recommendations included endorsement of state-wide meat inspection for meat and meat products not entering into interstate commerce, the inspection of poultry by the U. S. Department of Agriculture, the collection and publication of animal disease statistics, improved organizations for the control of live stock diseases, etc. The abstracts in the following pages give a comprehensive view of the program.

Preparation of Brucella Antigen[5]

In the light of our present knowledge of the variations which may occur in the sensitivity of Brucella antigens by growth on various media, dissociation, heat, preservatives, density, and the difference in antigenicity of various strains, it is easy to see why vast differences have occurred in the antigens produced by various laboratories. Many laboratories developed a procedure of antigen preparation and checked the results of serum reactions with cultural studies of infected animals with the result that a titer of 1:100 was adopted as the minimum titer denoting an infected animal. Although the 1:100 titer was accepted as an indication of infection, no standard method of antigen preparation was accepted. On comparative or check testing of various antigens, very divergent and confusing results were observed.

Much was done to clarify this situation by special committees of the Live Stock Sanitary Association and the A. V. M. A., but considerable variation was existent when the Federal-State coöperative program for the control of Bang's disease was inaugurated in 1934. The Bureau placed a representative in the field to visit the various laboratories and suggest changes tending toward uniformity, but a questionnaire sent to these laboratories in 1937 revealed, among others, the following conditions: Ten distinct types of culture medium, 60 different strains of Brucella (not all of which were *Br. abortus*), and nine different methods for determining the density were in use. As was to be expected, when these various antigens were check-tested against Bureau antigen, a wide discrepancy in sensitivity was observed in many of the products.

It has been apparent to the Bureau, as well as to all others concerned with Bang's disease testing, that the chief factor militating against the effectiveness of the program for the control of Bang's disease is the antigen used in the various states. On the recommendation of the conference on Bang's disease of the North-Central States, held in St. Paul in March, 1938, which was endorsed by the special committee on Bang's disease of the A. V. M. A. and other individuals and groups, the Bureau agreed to undertake the production of all antigen used in the Federal-State coöperative testing.



Adolph Eichhorn

In deciding upon what should constitute standard plate and tube antigens, studies had been made for several years of the procedures used by those laboratories whose products had been shown to give consistent results in Bang's disease eradication, and a standard for both types was developed.

Information furnished by the various states as to the amounts of antigen desired showed that 60,000,000cc of tube antigen and 360,000cc of plate antigen would be required annually. Obviously, such mass production necessitated extensive construction and provisions for washing, medium making, sterilization, seeding, incubation, harvesting, standardization, sterility testing, storage, and packing. The Bureau made the necessary preparations and began the production of antigen, taking every essential precaution to insure products not only pure from a bacteriological standpoint but uniform in agglutinability.

The Bureau is not in a position to supply the antigen other than in connection with the official testing program; but realizing the advantages of a uniform standard antigen for testing for brucellosis, it is willing to furnish a detailed method of preparation to state and private laboratories and to practitioners carrying out testing, and to undertake the standardization of antigens submitted from all institutions which are authorized to prepare test antigens.

[5] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by A. Eichhorn and H. I. Thaller, Animal Disease Station, Bureau of Animal Industry, U. S. Department of Agriculture, Beltsville, Md.

Problems Arising in Bang's Disease Control in the Western States[8]

A Bang's disease control program must be predicated upon scientific facts. In order for it to be successful it must also be practical. The cattle industry and the cattle owners, in particular, must be in a position to coöperate on a practical program that is not too expensive.

The present program of testing and slaughtering reactor cattle seems to be successful in some sections of the country. There are, however, differences of opinion with respect to what constitutes a proper control program. Problems which present themselves in the range areas make it difficult for both sanitary officials and live stock men to comply with all the requirements of the present program.

It is not possible, under range conditions, to test the range and semi-range cattle frequently enough to produce clean herds. It is impractical to attempt to keep tested animals apart from non-tested or infected animals under range conditions. In some sections dairy cattle also mingle with range cattle, both on the range and around the ranches and fields. Experience has shown that in some herds that have been freed from the disease by a test and slaughter program it has been impossible to keep them clean, and reinfection has been more disastrous than the initial infection.

Opinions differ with respect to a calfhood vaccination program. Experimental work so far is promising. Some herds have been brought to an approximate disease-free status by a program of calfhood vaccination in combination with a test and slaughter method. Calves vaccinated between four and eight months of age seem to develop a great deal of immunity. Those that react after a period of 18 months following vaccination are removed from the herd. Those that remain negative are added to the mature breeding herds. The mature animals that remain in the herd are tested annually, and where practical, semi-annually, and the reactors removed.

After following this practice over a period of a few years, the result is a herd composed of mature animals that have remained negative to the test, together with animals that have grown to maturity from the vaccinated calf herd that have remained negative to the test, which also carry some degree of immunity.

It seems that this type of herd is easier to develop and to keep free from infection under range conditions. This kind of program could be inaugurated in a range country because it is more practical and less expensive than the present program. Under our conditions it has so far seemed desirable. It produces the results we are seeking.

From our experience we have found that the pure-bred range cattle, particularly those in the breeding herds that are around the ranches, carry a higher percentage of infection than do the dairy cattle or the range cattle that spend most of their time on the open ranges. However, these animals mingle, at times of the year, on the ranges and around the ranches and fields. The lower percentage of infection in the cattle on the open ranges might more easily be controlled through a combination calfhood vaccination and testing program.

I cannot but feel that we could make some rather definite progress in our range areas with a program which would permit the vaccination of calves, under official supervision, in combination with a test and slaughter method. If it were possible to get accurate information from the various field experiments on calfhood vaccination being carried on in the states, the results would probably be enlightening. In the experimental work the federal government has conducted in our state we have not had an opportunity to check as closely as we would like. In some cases the cattle owners who coöperated could not keep an accurate record of some of the vaccinated calves, due to the fact that they were out on the range and, in some cases, were stolen or lost or died from other causes. The records that are available, however, indicate that the

[8] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by W. H. Hendricks, State Veterinarian, Salt Lake City, Utah.

program could be a very great aid in controlling the disease.

In 1934, seven herds were on the calfhood vaccination program. Ninety-one calves were vaccinated. Of these, 74 were tested in 1935, with the following results: 66 negative, six reactors, two suspects.

In 1936, 47 of the 74 were tested the second time. The results were 46 negative and one reactor.

In 1937, 46 calves were tested for the third time, 41 being negative and five suspects.

In 1938, 41 calves remained and were tested for the fourth time. Results: 39 negative and two suspects. Thirty-three of these calves were bled in 1939; all were negative to the test and have calved normally.

In 1935, eight new herds were put on the program, with 71 vaccinated calves. In 1936, 56 of these calves were tested with the result that 51 were negative and five suspects.

In 1937, 54 were tested the second time: 42 negative, four reactors, eight suspects.

In 1938, 31 were tested the third time with 29 negative, one reactor, one suspect. Thirty of these calves were bled in 1939; all were negative to the test.

In 1936, 16 new herds were put on the program and 103 calves were vaccinated. In 1937, 87 of these calves were tested: 75 negative, three reactors, nine suspects.

In 1938, 75 were tested the second time, with 66 negative, four reactors, five suspects. Sixty-two of these were tested in 1939, with the result that 57 were negative, two reactors, three suspects. All calved normally.

In 1937, 25 new herds were put on the program and 202 calves were vaccinated. In 1938, 158 of these were tested with 140 negative, three reactors, 15 suspects. In 1939, 149 of these were tested again, with the result that 130 were negative, 12 reacted, and seven were suspects. All calved normally.

■ The total number of calves vaccinated from 1934 to 1937 was 467 head. Of these 467 vaccinated calves 305 were tested in 1938 with the following results: 274 were negative, 8 reactors and 23 suspects. The percentage of vaccinated calves reacting was 2.6. These calves were in herds in which the infection was 15% or higher. In most cases we endeavored to keep controls in the herds also. The controls, in most instances, reacted and aborted. Very often they proved to be inferior animals and cattlemen disposed of them rather than keep them. Consequently we were unable to keep our records on them.

■ We have several range herds and some dairy herds that are apparently free from Bang's disease, as a result of calfhood vaccination in combination with a test and slaughter program. One herd in particular is a pure bred range herd. Before the Bang program was started on this herd in 1934, breeding troubles were frequent and many animals consequently aborted. Several of the mature animals developed swollen joints and spoiled udders. It was decided to test the mature animals in this herd and eliminate those that reacted. In addition to this procedure we vaccinated the calves in conformity with the present calfhood vaccination program. It was a range herd and it was difficult to always have the animals available for testing when we needed them. But we felt that by vaccinating the calves between the ages of four and eight months and testing them after a period of 18 months had elapsed, that we could build a negative herd that would carry some degree of immunity. The calves that reacted to the test after the period of 18 months following vaccination were removed from the herd as reactors. The mature animals in the herd were tested periodically and those that reacted were removed from the herd. Today the breeding herd is composed of cows that were tested as mature animals and remained in the herd as negative, together with calves that were vaccinated each year and continued to give a negative reaction to the test. This herd, as I stated, had, at one time, a high percentage of infection. Recent tests on this herd disclose only one suspect. The herd is having no breeding troubles. All are calving normally and there are no complications developing in the herd. The herd is on the show circuit and probably has as much or more opportunity to come in contact with infection than do other herds. It seems that the disease free status of this herd, together with the immunity developed, is of considerable value. The owner is well pleased with the results and has been able to stay in the cattle business, whereas, under a testing and slaughter program he would have been forced out of the business.

We have a number of other herds that have been freed from Bang's disease with this same method. This program, in the opinion of some, may not be desirable from the standpoint of ultimately freeing the herd from infection and controlling the disease. Yet, our experience indicates that there is a possibility that it does just that. Such a program is practical and feasible under our range conditions. We believe that we could get the coöperation of the cattlemen on such a program because it would be possible to vaccinate the calves and it would also be possible to test the mature animals, particularly the infected herds, at least once a year so that we could gradually move the infected mature animals out and add to the breeding herds the calves that were vaccinated and remain negative to the test. These herds that have been handled in this manner have, in our experience, remained clean while herds that were tested under the test and slaughter program, and attained a negative status, have later developed a serious reinfection, due to their susceptibility. We believe that if we could develop a program in our western range areas whereby such herds as may be deemed advisable by the supervising officials, could be placed under a program of calfhood vaccination in combination with a test and slaughter program on mature animals, that we could get results. The vaccinated calves would be tested also, after a period of 18 months had elapsed and those that reacted would be moved out of the herd in the same manner as reactors in the mature herd and these reactors would be eligible for the Federal and State indemnity payment. If there is anything to the calfhood vaccination program, and I believe there is, then it would be a distinct aid in our range areas in controlling the disease. It will overcome many of the objections raised by our cattlemen. It will permit the cattleman to stay in the business while getting a mature herd free from disease and at the same time will provide him with a new herd composed of animals that have some immunity to infection.

The Present Status of the Federal-State Bang's Disease Program[12]

There was a considerable increase in the number of herds and cattle under supervision in the federal-state Bang's disease project over the number so reported a year ago, the increase in the number of herds being approximately 30% and in the number of cattle about 14%. On November 1, 1939, approximately 21% of the dairy and breeding cattle over six months of age in the United States were under supervision.

During the fiscal year ended June 30, 1939, agglutination blood tests were applied to approximately 7,600,000 cattle, located in about 725,000 herds. Of the cattle tested, 219,165, or about 3%, were classified as reactors. Many of these tests were retests.

The average federal payment made to owners of reacting cattle is, at this time, approximately $15.50, the state payment about $17.65, and the salvage about $34.00. The average appraisal is about $93.00, and approximately 11% of the reactors are registered purebred cattle.

In 24 states the initial testing of all the dairy and breeding cattle over six months of age has been or is being conducted in about 490 counties, up to November 1 of this year, and in about 400 of these counties in 19 states the initial tests have been completed, showing an average cattle infection of 2.7% and a herd infection of 5.2%.

All the antigen now being used in official work for Bang's disease is being furnished by the Bureau of Animal Industry and has been since about July 1 of this year. This arrangement has proved very satisfactory, and plans have been made to have all the antigen for private use prepared according to the methods followed in making the Bureau antigen.

Further studies have been made on the subject of calfhood vaccination against brucellosis in the field, and work is being done in about 260 herds in 24 states. In these herds there was more than 15% infec-

[12] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by A. E. Wight, Chief of the Tuberculosis Eradication Division, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

tion when the work was taken up in January, 1936. Since that time approximately 13,000 calves between the ages of five and seven months have been immunized, but

Benjamin Schwartz

the work has thus far not progressed to a point where it is possible to formulate any final conclusions regarding this practice.

No federal payment can be made in excess of the amount paid by the cooperating state or other agency for Bang's disease reactors since May 1, 1939; local funds have been provided to the extent of approximately $3,500,000 in 35 different states.

Results of the work thus far show that good progress has been made. There are about 55,000 state certified herds, containing about 1,100,000 cattle, and there are in the neighborhood of 200 counties in 15 states where it has been demonstrated by actual testing of all the cattle that Bang's disease does not exceed 1% of the cattle population and exists in not more than 5% of the herds of cattle in those counties. As a result of much individual herd work and the removal of reactors, the disease has been eradicated from a large number of herds of cattle throughout the country and thereby has reduced the incidence of the disease among cattle as it is estimated on a country-wide basis.

Parasitic Diseases[2]

Research into the parasitic diseases of animals by the Zoological Division of the federal Bureau of Animal Industry during the past year included (1) skin tests for trichinosis, (2) phenothiazine as an anthelmintic, and (3) barium antimonyl tartrate as a treatment for the poultry gapeworm.

In an endeavor to find an antigen that can be used for an allergic skin test of trichinosis in swine, 55 different antigens were experimented with on approximately 1,500 swine. Some of these swine were experimental animals artificially infected with *Trichinella spiralis* and others were market swine tested at packing houses before slaughter.

Notwithstanding that others have reported success with a skin test for the detection of trichinosis in rabbits and man, none of the 55 antigens tested in this experiment gave satisfactory results. In each test some uninfected animals (as determined by post-mortem examination) reacted, and some infected animals failed to react positively to the test.

The Committee state that the possibilities have been by no means exhausted with the test of the 55 antigens, and express the hope, based upon the experiments with these antigens, that one may yet be found that will enable veterinarians to detect the presence of trichinosis in swine, if not by a test on the living animal, by injecting the antigen shortly before the animals are slaughtered and reading the reaction at slaughtering, thus obviating the tedious examination of sections of the diaphragm.

■ Experiments with phenothiazine established its efficacy in removing internal parasites from swine and sheep; in the latter species it was found to remove a large proportion of the hookworms, nodular worms, both species of stomach worms, and intestinal trichostrongyles. Further, the therapeutic dose of phenothiazine is only a fraction of its toxic dose. Commercial pheno-

[2] Abstract of the report of the Committee on Parasitic Diseases (Chairman: Benjamin Schwartz, Chief of the Zoölogical Division of the Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.) at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939.

thiazine (prepared for use as an insecticide and containing a conditioner for that purpose), was found to be highly efficacious[3]; the recrystallized product was found to be even more efficacious as well as easier to administer (see the report by Habermann and Harwood on pages 24-29 of this issue). A limited series of tests with crude phenothiazine, which does not contain the conditioner placed in insecticidal phenothiazine, indicated that this substance is satisfactory as an anthelmintic, with the advantage of being cheaper than the recrystallized product.

■ It was demonstrated that powdered barium antimonyl tartrate, when inhaled by affected birds, is an effective drug for the treatment of gapeworm infestations in young chickens and turkey poults. The birds are placed in a closed container and exposed to the chemical for 15 or 20 minutes. For a box with a capacity of eight cubic feet (which will accommodate about 50 chicks or poults), one ounce of the fine powder is sufficient. The box should be deep enough to permit the birds to stand erect, and should be about two-thirds filled with the birds. One-third of the powder is introduced by means of a dust gun through an opening in one side of the box, and the box is tilted slowly from side to side, the movement encouraging dispersal of the powder and deeper breathing by the birds. Five minutes later the second third of the powder is introduced and the tilting repeated. The remaining powder is introduced in the same manner five minutes later, and the birds are left in the box for another five minutes. Tests showed the average efficacy of this treatment for the removal of gapeworms to be 92% for chicks and 96% for turkey poults. Experiments are now in progress to determine the efficacy of this drug for the removal of gapeworms from mature turkeys.

Barium antimonyl tartrate is not yet available commercially. It may be prepared as follows: For approximately 1¾ pounds of the drug, dissolve 244.3gm of crystallized barium chloride in 800cc of distilled water at a temperature of 70° to 75°C., and pour this solution slowly into a warm solution of crystallized potassium antimonyl tartrate (667.8gm to 1,700cc of distilled water), stirring vigorously with a glass rod. The precipitate that forms is filtered at once through paper with a suction filter, washed with cold water and then with alcohol to facilitate drying. The drying is carried out at room temperature, since the higher temperatures while the chemical is wet may cause decomposition. When properly prepared the product will pass through a 100-mesh sieve; otherwise it must be ground to that degree of fineness.

[3] *Vet. Med.* 34:7, pp. 440-443.

Swine Enteritis in Veterinary Practice[14]

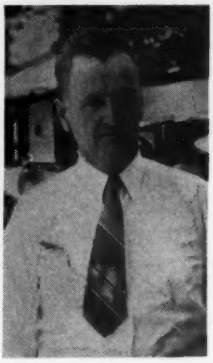
Swine enteritis is related to faulty nutrition, production mismanagement, and infected, insanitary premises. These are visible and tangible factors which practitioners may point out to owners and which constitute the first line of attack for the combined forces of the owner and veterinarian.

Among the different forms of enteritis, the more common ones are those affecting baby pigs, due to improper feeding of the dam; necrotic enteritis of weanling pigs, usually found on insanitary premises and also bearing evidence of vitamin B deficiency; and infectious hemorrhagic enteritis, most commonly found in commercial

[14] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by John B. Bryant, Mount Vernon, Ia.

John B. Bryant

cattle and swine feeding establishments, for which owners and veterinarians are anxiously waiting for assistance. Enteritis is a serious menace to serum-virus reactions. Cases are reported of hogs reared on



L. P. Doyle

clean pastures, removed to infected premises, and vaccinated, with resultant heavy losses.

Nonspecific enteritis is produced by various irritants. Cases have been reported that were due to sand consumed by baby pigs, pebbles consumed by shotes, a boar succumbed to enteritis from feeding on the carcasses of baby pigs, and a nostrum peddler killed 50% of a drove with his powder.

Fundamentally important factors in the incidence of enteritis are: first, the changed character of feeds, due to soil depletion and the natural leaching processes, also the feeding of by-products from the industries and the general lack of knowledge concerning these changes; second, forced production performance, for a quick turn-over of capital, has given rise to early forcing of immature pigs whose digestive tracts are unable to withstand repeated slight attacks of indigestion due to feeds lacking nutritional balance and completeness, and, third, concentration of infection, which is the result of increased production and crowding swine in small lots and pastures without regard to proper sanitation. Such conditions favor parasitic infestations with their accompanying nutritional, toxic and irritating effects which predispose to bacterial invasions and enteritis.

Infectious Types of Swine Enteritis[1]

The fact that hog cholera frequently affects the intestinal tract probably accounts for a good deal of the confusion regarding enteritis in swine. Experience has taught many practicing veterinarians and laboratory workers to be cautious in eliminating hog cholera from consideration as a cause of swine enteritis. An attempt at classifying forms of enteritis in swine does not seem advisable at this time.

Coccidiosis does not appear to be nearly as important in swine as it is in other species of live stock. *Salmonella suipestifer*, when fed in cultures, has been found to cause enteritis to a limited extent. However, there is still need for critical examination of the evidence as to how important this microörganism is as a primary cause of self-perpetuating enteritis in swine.

Swine dysentery, called by such common names as bloody diarrhea, bloody or black scours and bloody flux, is definitely a self-perpetuating disease of the intestinal tract. Its clinical features has probably caused it to be mistaken for coccidiosis. The pathology of dysentery is essentially that of a colitis, frequently accompanied by a gastritis. A striking pathologic feature of the disease is the great extent to which the colon may be affected, while the small intestine appears normal. Dysentery has an established place among the destructive diseases of swine. The causative agent is present in the colon content and in the bowel discharge of affected hogs.

Salmonella suipestifer has been found in a large portion of certain groups of pigs affected by dysentery. However, typical dysentery has not been reproduced by feeding cultures of *S. suipestifer*. Dysentery, then, appears to be similar to cholera in that the mere finding of *S. suipestifer* leaves unanswered the question of what was primarily wrong with the hog.

For the control of swine dysentery there is sore need of effective control of traffic in infected swine.

[1]Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by L. P. Doyle, Department of Veterinary Science, Purdue University, Lafayette, Ind.

Swine Enteritis[6]

We have encountered many cases of "enteritis" in our work during the year that have been very interesting to study, and very baffling in attempts at control. It is my observation from cases seen and those reported by veterinary practitioners that this condition has caused greater losses in western Iowa during 1939 than hog cholera and all other swine diseases combined during the past six or eight months.

The condition encountered so extensively this year has been often a necrotic colitis, or gastrocolitis. Very few herds so affected have shown involvement of the small intestine, such as we formerly encountered years ago and termed "necro."

In the majority of cases the colitis was accompanied by a bloody discharge from the cecum, colon, and rectum. Many of these cases were so acute that hemorrhages did not have time to appear; the pig, shote, or hog would eat and then fall over and die at the trough. The owner in many such cases had not noticed anything wrong with that individual pig.

The condition strikes herds kept on "new" ground and under good sanitary conditions just as in herds maintained in filthy surroundings. It has hit herds of purebred shotes, small herds receiving almost individual attention, just as it has hit herds badly neglected. It strikes at herds being fed in apparently a proper manner and other herds where the feeding ration is open to serious question. It attacks impartially herds that have had no serum and virus and those vaccinated. When hog cholera virus is administered to herds already infected with "enteritis" the results are usually disastrous. But it strikes herds long after vaccination, just as it does herds never so treated.

In short, losses have been heavy in all classes and groups of swine and under all conditions.

Treatment has been disappointing. Alkaline solutions are used extensively and must be of some value. However, the farmers have the notion that "alkaline solution" simply means "lye," and much inhumane treatment of sick hogs has resulted.

John S. Koen

None of the laboratories have been able to establish the cause of the trouble, or to prescribe a treatment that is consistently successful.

There are two outstanding herds in Iowa that show, convincingly, the value of a swine sanitation program. While neither of them follow the McLean County system of swine sanitation, they have developed a system of their own based on the McLean County system.

One herd, the largest of which I have any knowledge in Iowa, is that of Johnson Brothers, northeast of Laurens. Nearly 1,400 spring pigs were vaccinated in this herd the latter part of August. For 10 years they have avoided "enteritis," "barnyard infection," etc., until the herd is the largest in western Iowa. A 10-year successful experience proves the value of swine sanitation.

The other herd was raised by John Hendricks, near West Liberty. Ninety-two spring pigs were raised on concrete, never being on ground from time of farrowing until going to market the latter part of August. By cleaning the concrete slab between each two feedings, these pigs never had a chance to pick up infection. Clean feed and clean water, plus clean quarters, enabled Mr. Hendricks to market these hogs at six months of age averaging 243 pounds.

[6] Abstract of a discussion presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by John S. Koen, Assistant Veterinarian, Bureau of Animal Industry, U. S. Department of Agriculture, Storm Lake, Ia.

The General Tuberculosis Eradication[11]

During the period from December 1, 1938, to November 1, 1939, eight of the remaining 14 counties in the State of California not in the modified accredited area were placed in that status. During the progress of the nation-wide campaign, inaugurated in 1917, a total of more than 220,000,000 tuberculin tests have been applied, resulting in the removal of approximately 3,750,000 reactors.

Public interest has continued in the tuberculosis eradication work. During the fiscal year ended June 30, 1939, tuberculin tests were applied to approximately 11,185,000 cattle, located in about 750,000 herds. Approximately 60,000 or 0.5%, reactors were located, which is the lowest degree of infection reported since the inauguration of the campaign. Of these reactors, approximately 45% were in California.

While particular emphasis has been placed on the eradication of tuberculosis from cattle, a considerable amount of attention has also been given the problem of avian tuberculosis. This disease affects poultry and swine, and is the cause of considerable loss each year. During the past fiscal year approximately 10,000 flocks, located in 10 states, were observed by veterinarians assigned to that project, and, as a result of these inspections, the presence of infection was determined on approximately 1,100 farms. Veterinarians engaged in the tuberculin testing of cattle continued to observe poultry flocks in states where it is deemed advisable to do so, and during the past fiscal year they observed approximately 98,000 flocks, in 3,300 of which infection was reported.

The eradication of tuberculosis in live stock continues to show gratifying results in the reduction of this disease in swine. Federal meat inspection records indicate that during the past fiscal year 8.8% of all swine slaughtered at establishments operating under Federal supervision showed some evidence of tuberculosis. This is a slight decrease in comparison with the previous year. Reports from packing houses are very helpful in locating centers of infection of tuberculosis when the disease is found on post-mortem examination of cattle and hogs.

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The Pathogenicity for Cattle of the Avian Tubercle Bacillus[15]

The bovine form of the tubercle bacillus was and in all probability still is the infective agent responsible for most instances of tuberculosis in cattle. With the elimination of the frankly tuberculous animals, difficulty in demonstrating at post mortem definite lesions of tuberculosis in cattle that have reacted to the tuberculin test is becoming more difficult. Avian tubercle bacilli do occasionally infect cattle, and in regions where opportunity for infection with avian tubercle bacilli exists this organism should be given serious consideration in explaining slight, nonprogressive lesions and sensitivity to tuberculin, providing infection with the bovine type of the organism is unlikely. The exact status of this problem has by no means been established.

Occasionally cattle sensitized under natural conditions with avian tubercle bacilli will react rather typically to mammalian tuberculin, and also, it seems not unlikely that in the United States the avian tubercle bacillus sensitizes more cattle to mammalian tuberculin than is generally recognized. Furthermore, it is possible that at least some of the so-called nonvisible lesion reactors react to mammalian tuberculin as a consequence of sensitivity induced by avian tubercle bacilli.

While the lesions produced in cattle by the avian tubercle bacillus are sufficiently characteristic to be acceptable as presumptive evidence of a tuberculous infection, there exist no features that would definitely

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distinguish the lesions as specifically those due to the avian tubercle bacillus. The morbid changes produced in cattle by the avian type of the organism are inclined to remain localized. No cases of destructive, widespread or generalized tuberculosis in cattle due to the avian tubercle bacillus have been reported.

The evidence reviewed indicates that interpretation of the tuberculin test in cattle is made more difficult and uncertain by the existence of tuberculosis in poultry. A definite and positive program for the elimination of tuberculosis in fowl should constitute the next phase of our attack on tuberculosis in animals.

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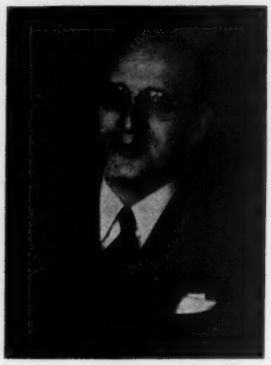
Reaccreditation of Modified Accredited Counties in the State of New York[4]

In New York, initial tuberculin tests and subsequent tests until the herd is accredited, are made by whole-time federal, state or county veterinarians, but all tests for reaccrediting herds are made by practitioners. The herds in each county are allotted among the practitioners in that county, each practitioner being assigned one or more townships in which he is to be responsible for reaccrediting all the herds. They are paid for the tests at $2.50 per herd plus 20c for each animal in the herd. Thus, for testing a one-animal "herd," the practitioner receives $2.70; for testing a 100-animal herd, $22.50. On this basis, the average cost for each animal tested is 32c. Fees for testing are paid by the state, which appropriates $450,000 annually for this purpose. Approximately 400 practitioners participate in the work, which gives them an average annual income from this source of $1,125.

It is the desire of the state Bureau of Animal Industry to test all herds in the state annually until there are no more reactors, but the $450,000 appropriated annually for this purpose by the state is not sufficient for testing a[illegible] herds in the state, but only for about [illegible] herds. Therefore, the herds have been [illegible]ided into three



E. T. Faulder

groups. In 14 intensive dairy counties, where 38% to 53% of the animals reacted on the initial test, all herds containing more than eight animals, and those smaller herds in which reactors have been found, are tested annually. Herds containing eight animals or less, in which there have been no reactors on the initial or any subsequent test, are tested for reaccreditation triennially. In the remainder of the state, retesting of accredited herds is on a two-year basis. When infection is found in a herd, the testing is taken over by whole-time state or county veterinarians until the herd is again accredited, when it is returned to the local practitioner for the reaccrediting tests.

There are approximately 50 whole-time country veterinarians in New York State. County boards of supervisors have appropriated $3,000,000 for the employment of county veterinarians, assistant county veterinarians, and office help since 1921.

Since the tuberculosis eradication project was initiated in New York in May, 1918, the state has spent $47,000,000 and the federal government $11,000,000 for the payment of indemnities for reacting animals. This is exclusive of operating costs.

[4] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by E. T. Faulder, Director of the Bureau of Animal Industry, New York State Department of Agriculture and Markets, Albany, N. Y.

Infectious Equine Encephalomyelitis in 1939[18]

Continuing the program begun in 1935, the federal Bureau of Animal Industry compiled data on the incidence, mortality, and other aspects of infectious equine encephalomyelitis during 1939. During the five years considerable variation in intensity of the disease has been experienced. In 1935, 23,512 cases were reported; in 1936 there were only 3,929; in 1937 and 1938 there were 173,889 and 184,662, respectively. In 1939 a distinct drop in incidence occurred; 7,988 cases have been reported from 42 states, the six states reporting no disease being Mississippi, New Hampshire, Pennsylvania, South Carolina, Tennessee, and West Virginia.

The rate of incidence for the entire country in 1939 is calculated from present figures to be 1.12 cases per 1,000 animals; the case mortality as 30.3%. As in previous years, the bulk of the cases, 7,136, were reported from 28 states west of the Appalachian Mountains, less than 600 being reported from Eastern states. The rate of incidence in the former area was 1.07 cases per 1,000 and for the latter, 2.32 per 1,000. The mortality in the West was 26.7% and 85.6% in the East.

The reports received this year show a total of 805,247 horses and mules vaccinated with formolized chick-embryo vaccine in areas where infectious encephalomyelitis appeared. Of these, 289 are reported to have developed encephalomyelitis. The reported incidence in vaccinated animals was 0.36 cases per 1,000, as contrasted with 1.3 cases per 1,000 in unvaccinated animals in the same states.

The Division of Virus-Serum Control reports 9,462,305 10cc doses produced in 1939. Of these, it may be fairly estimated from all the information at hand that about 6,000,000 doses (3,000,000 2-dose vaccinations) were administered. These figures are almost four times greater than the number actually reported to the Bureau. Most of the states reporting indicate that their figures are incomplete and it is probable that the estimate given above more nearly represents the actual number of horses and mules vaccinated.

Harry W. Schoening

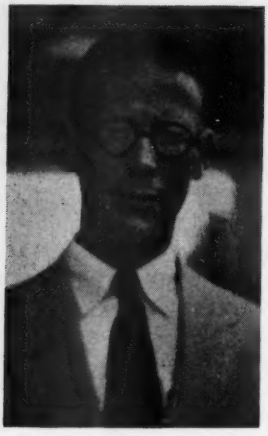
Vaccination of itself has apparently been responsible for the death of some few animals, not from encephalomyelitis, but rather from the procedure *per se*. Reports from all sources indicate that severe local reactions or untoward systemic reactions were of comparatively minor general importance in encephalomyelitis vaccination. The Army vaccinated over 35,000 animals without any serious reactions. There was not a single loss from encephalomyelitis in these animals. However, in a few instances, the experiences of individual practicing veterinarians were reported as unfavorable. In these cases there were reported gross edema, with or without other symptoms of infection, and abscess formation. Tests conducted by the distributing firms themselves, the Bureau, and other agencies showed that the vaccines used were free of contaminating bacteria. The reports as a whole indicate that many have not anticipated the slight to moderate swellings to be expected when formolized tissue is injected.

Relatively very few cases of anaphylactic shock, with an occasional death, were reported. Inappetence, dullness, and drowsiness, and even temporary weakness and incoördination, were reported following vaccination in a few cases. Symptoms of acute colic as well as abortion were encountered in a few instances.

[18] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by H. W. Schoening, Chief, L. T. Giltner and M. S. Shahan, Pathological Division, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Equine Encephalomyelitis in Avian Hosts[7]

In three outbreaks of encephalomyelitis in pheasants diagnosis was made by the inoculation of embryonated eggs with brain



F. R. Beaudette

filtrates. The virus was neutralized by immune eastern type serum as determined by egg inoculations. In all cases there was the usual history of paralysis of the legs, ataxia, etc. The affected flocks were located at Forked River, Freehold and Sparta, N. J., and represent three counties. In two of these counties the disease in horses is not uncommon. The outbreaks began in late summer and in two cases continued after killing frosts. The incidence of the disease in the flock and the mortality were high. The disappearance of vegetation in the holding pens was followed by picking, which may be a factor in the spread of the disease. Gapeworm infestations also appeared in the history of most outbreaks.

Other pheasant flocks also may have been affected by the disease. One of the flocks in which the disease was diagnosed probably had the disease the previous year, and in another flock this year's outbreak probably represents the third consecutive yearly outbreak.

[7] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by F. R. Beaudette, Poultry Pathologist, New Jersey Agricultural Experiment Station, New Brunswick, N. J.

Fundamentals of Animal Disease Control[10]

The live stock sanitary setup in most of the states was organized in the last decade of the 19th century or the first decade of the present century, *i.e.*, 30 to 50 years ago. New information on the importance of certain animal diseases to the public health, changes in animal production and in the distribution and relative importance of different diseases, in methods of transporting animals, in the availability of competent veterinarians, and the developments of research amply justify a reëxamination of the live stock sanitary services of the states with a view to better adapting them to present conditions and to making such improvements in them as experience has shown to be desirable.

The United States Live Stock Sanitary Association is the logical organization to initiate and promote improvements of the animal disease control service in the various states. It is unofficial and thus possesses complete freedom of action. It is interested in the animal disease situation in each of the states. It is a scientific organization obligated to serve only the best interests of the live stock industry of the whole country and its membership possess the largest pool of information and experience in the control of animal disease to be found anywhere in the nation.

The plan of organization of a live stock sanitary service suggested would consist of:

1. A live stock commission of five members—three live stock men and two veterinary practitioners.
2. A state veterinarian.
3. A state diagnostic laboratory.
4. One or more whole-time assistant state veterinarians.
5. All the qualified veterinary practitioners in the state. The state veterinarian and assistants and the laboratory employes would be under civil service regulations.

Some of the advantages claimed for this plan are economy, efficiency, and freedom from political influence.

[10] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by D. M. Campbell, Editor of *Veterinary Medicine*, Chicago, Ill.

State-Wide Meat Inspection

An eminent authority once said that on no question of social economy is the general public more ignorant than on that of the wholesomeness, or otherwise, of the flesh they consume. Therefore, it is important that sanitary authorities guard them against abuse and injury from this source by a proper system of meat inspection. There are a number of infectious diseases of animals which are communicable to man; even if this were not so, considerations of esthetics and common decency would require the inspection of animal products.

It is a common practice, when animals become unfit for any other purpose and there seems to be a danger of losing their monetary value, to slaughter them for meat. This practice, with its attendant dangers, will continue until the inspection of all meat stops it.

The federal system of meat inspection is limited to interstate shipments. Most cities have local ordinances protecting the meat supply of their inhabitants. But, by and large, meat hygiene is lacking for the smaller towns and the rural population.

The obvious remedy for this situation is state meat inspection, similar to the federal inspection of meat that goes into interstate commerce or for export. That such a measure is practicable is evidenced by the experience of Connecticut, where, since the adoption and enforcement of statutes providing for a system of state meat inspection and hygiene, the greatest improvement has been noticed in the handling of meat products.

That there has not been a more general adoption by the various states of such a system of meat inspection is due chiefly to the opposition of those interested from a monetary point of view in maintaining the traffic in uninspected meat. It would appear desirable to impress upon our state legislators the selfishness of this opposition, and to urge them to consider the health of the multitude, rather than to favor a few who have no scruples for the welfare of others.

[9] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by George E. Corwin, Deputy Commissioner on Domestic Animals, Hartford, Conn.

Rabies in the United States[16]

Through the Bureau of Animal Industry, information was made available from a questionnaire sent to the various state live stock sanitary officials and state health officers concerning points in connection with the control of rabies in their states. The questionnaire concerned itself principally with the authority in the state charged with the control of the disease, the responsibility for its control, the laws and regulations under which the authority acts, the method of enforcement, the source of funds, and the number of cases of rabies for the year 1938. There were reported for the United States 8,452 cases in dogs, 413 in cattle, 32 in horses, 164 in sheep, 42 in swine, 207 in cats, 11 in goats, 44 in miscellaneous animals, and 47 in man, making a grand total of 9,412 cases.

* * * *

Turkey Disease Control[17]

In commercial turkey flocks, general preventive measures include the selection of strong, disease-free breeding stock; proper conditions of ventilation, temperature, humidity, etc.; strict sanitation; the feeding of a complete, balanced ration; and systematic culling. In addition to these general measures, the following have proved valuable in controlling specific diseases: For infectious enterohepatitis and trichomoniasis, it has been found that phenylmercuric nitrate, given in the drinking water in a dilution of 1:50,000 every third day has shown the most promise. For coccidiosis, the best results have been obtained by putting the poults on a dry mash feed containing 40% dried skimmilk or buttermilk for five to seven days. Sinusitis may be treated by irrigating the affected sinuses with a mild sodium bicarbonate solution, then injecting about two drops of an ephedrinated inhalant, or two drops of a 5% silver nitrate solution.

[16] Abstract of the report of the Committee on Rabies (Chairman: H. W. Schoening, Chief, Pathological Division, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.), presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939.

[17] Abstract of a paper presented at the 43rd annual meeting of the U. S. L. S. S. A., Dec. 6, 7, 8, 1939, by L. D. Frederick, Chief Veterinarian, Swift & Company, Chicago, Ill.

The Effects of Injection into Bovidae of Emulsions of Anaplasma-Infected Ticks

By CHARLES W. REES, Washington, D. C.
Bureau of Animal Industry,
U. S. Department of Agriculture

THE object of the experiments reported in this paper was to determine whether injection, into susceptible Bovidæ, of emulsions of Anaplasma-infected ticks would protect them against attacks of anaplasmosis.

Emulsions were prepared from three species of ticks, namely, *Boöphilus annulatus*, *B. annulatus* var. *australis*, and *Rhipicephalus sanguineus*. The two species of Boöphilus were obtained from Texas and Florida through the Division of Tick Eradication and Special Diseases of the Bureau of Animal Industry, and *R. sanguineus* from a strain reared in the laboratory of the Zoölogical Division at Beltsville, Md. The first Boöphilus emulsion was prepared by grinding in a mortar in physiologic saline solution six engorged female ticks from Texas and 31 from Florida; the ground-up materials were mixed and the débris removed by filtration through ordinary filter paper. The second Boöphilus emulsion was prepared from larvæ of both lots of ticks after they had engorged on a susceptible bull for 72 hours; 5gm of this material was used. The reason for permitting the larvæ to engorge before making the emulsion was that Theiler and Du Toit[1] reported that emulsions of infected ticks, that had previously engorged for 72 hours, transmitted heart-water and theileriosis, but that emulsions of unengorged ticks failed, as a rule, to transmit either of these diseases. Two other Boöphilus emulsions were prepared by filtering the emulsions made from unengorged and engorged ticks through a Seitz filter. Inoculation of some of these filtrates into fermentation tubes, nutrient broth and nutrient agar showed that the filtrates were sterile. The Rhipicephalus emulsion was prepared by grinding, as described above, five engorged nymphs and filtering the resulting emulsion through filter paper.

TABLE 1.—Results of Vaccination of Bovidæ with Emulsions of Ticks

Animal	Species of Ticks*	Infections Carried†	Kind of Emulsion‡	Date of Injection	Date of Exposure to Anaplasmosis	Date of Reaction to Anaplasmosis
Bull 112	*B. a. a.* *B. a.*	*Bab. arg.* *A. m.*	f.p., u.t.	11-15-33		
	B. a. *B. a. a.*	*Bab. arg.* *A. m.*	f.p., e.t.	1-2-34		
	R. s.	*A. m.*	f.p., u.t.	3-8-34	4-15-34	5-17-34
Heifer 117	*B. a.* *B. a. a.*	*Bab. arg.* *A. m.*	s.f., u.t.	11-15-33		
	B. a. *B. a. a.*	*Bab. arg.* *A. m.*	s.f., e.t.	1-2-34	5-17-34	6-11-34

B. a. = *Boophilus annulatus*; *B. a. a.* = *Boophilus annulatus* var. *australis*; *R. s.* = *Rhipicephalus sanguineus*.
†*Bab. arg.* = *Babesia argentina*; *A. m.* = *Anaplasma marginale* (infections assumed as explained in text).
‡f.p. = paper filtrate; s.f. = Seitz filtrate; u. t. = unengorged ticks; e.t. = engorged ticks.

In order to demonstrate that the ticks were infected with Anaplasma some of them engorged on susceptible cattle under the following conditions: February 28, 1934, some larvæ hatched from eggs of *B. annulatus* and *B. annulatus* var. *australis* were mixed together in a cloth bag and confined to the scrotum of bull 115. On March 9 the bull came down with an in-

[1] Theiler, A., and P. J. Du Toit, 1928. The Transmission of Tickborne Diseases by Intrajugular Injection of the Emulsified Intermediary Host Itself. 13-14 Rep. Director Vet. Educ. & Research, Dept. Agric., Union of South Africa, Pretoria, Pt. 1, pp. 17-44.

fection of *Babesia argentina*, dying March 10. There was thus no opportunity to show that the ticks were infected with *Anaplasma marginale*. However, it was pointed out by Smith and Kilborne[2] that cattle that recovered from hemoglobinuric fever came down with what they regarded as relapse attacks of piroplasmosis, but Theiler[3] demonstrated that the former investigators were dealing with anaplasmosis. Unpublished data by the present writer show that during 10 years every non-fatal case of piroplasmosis was followed by an attack of anaplasmosis. Therefore, in Table I the two kinds of Boöphilus are reported as infected with the causative organism of both anaplasmosis and piroplasmosis. March 28, 1934, some nymphs of *R. sanguineus*, from the same lot as those from which the emulsion was prepared, engorged on bull 119. May 7, the bull showed symptoms of anaplasmosis, which proved that the ticks were infected with *Anaplasma marginale*.

The experimental data recorded in Table I show that both animals that were vaccinated with the tick emulsions came down with anaplasmosis following injections with virulent blood. Therefore, injections of emulsions did not transmit anaplasmosis, nor did they protect the animals from clinical attacks of the disease induced by the injection of virulent blood.

[2] Smith, T., and F. E. Kilborne, 1893. Investigations into the Nature, Causation, and Prevention of Texas or Southern Cattle Fever. Bul., 1 Bur. Anim. Indus., U. S. Dept. Agr., Wash., pp. 1-301.

[3] Theiler, A., 1910. *Anaplasma marginale* (Gen. and Spec. Nov.). The Marginal Points in the Blood of Cattle Suffering from a Specific Disease. Rept. Govt. Vet. Bacteriol., Transvaal Dept. Agr., 1908-9, pp. 7-64.

SIR ARNOLD THEILER—1867-1936

Sir Arnold Theiler, a native of Switzerland, spent more than 30 years in the employ of successive South African Governments. He was pre-eminent in investigation of the diseases of animals in warm climates, among other investigations that of anaplasmosis referred to by Doctor Rees. His work that attracted the widest attention in America was the investigation of bone chewing by cattle on certain areas of the velt. These investigations led to the recognition of phosphorus deficiency in dairy cattle in various districts of the United States, and a recognition of the cause of loin disease in the Southwest. He was the first to receive the medal of the South African Association for the Advancement of Science. Other awards included the Scott Medal of the African Biological Society and the Gold Medal of the Society of Exotic Pathologists of Paris. At the meeting of the 12th International Veterinary Congress in New York in 1934 he was awarded the Budapest Medal.

Pronounced Allotrophagia in Oxen

Subject.—Hereford oxen aged about two years.

History.—While I was stationed at Grootfontein School of Agriculture, Middleburg, Cape, a farmer reported that one of his oxen had died and that a few others appeared sick from a cause unknown.

Diagnosis.—Post-mortem examination revealed the rumen and reticulum almost completely filled with rounded river stones, the largest of which were about the size of a golf ball. On palpation of the abdomens of the sick oxen, which were all pot-bellied, it was evident they also had been ingesting stones freely.

Etiology.—Aphosphorosis possibly caused the craving, because the animals subsisted entirely on the veld.

Treatment.—I performed rumenotomy on one of the animals and removed 62 pounds of stones. First-intention healing took place, but, although the animal fed fairly well, it was still in poor condition when about two months later it was accidentally killed.—*O. T. De Villiers*, in the *Jnl. of the South African Vet. Med. Assn.* 10:3.

Chronic Equine Pyometra

September 27, 1939, a six-year-old piebald saddle mare was presented at the veterinary clinic of Kansas State College for treatment.

History.—The mare had foaled in June, 1938. There was dystocia, and a veterinarian was in attendance. The fetus was in anterior longitudinal presentation, with the forelegs retained and the hind legs extended under the body. The vagina and cervix were lacerated during the necessary embryotomy; the mare, however, apparently made an uneventful recovery, except for the fact that she did not come in heat and was not bred again.

In May, 1939, the mare started losing flesh, but her appetite remained normal, and two weeks later she seemed to improve. On about August 15 she started losing flesh again, with gradual distention of the abomen.

Symptoms.—There was pronounced distention of the abdomen, with a very noticeable dyspnea. Manual examination of the genital organs revealed that the uterus was greatly distended, ballooning back into the pelvic cavity along the right side of the vagina and causing a bulging of the anus. Adhesions had formed in the vagina, completely occluding the cervix, and a stricture of the vulva nearly closed the vagina. Adhesions had also apparently formed between the walls of the vagina and uterus where they were in contact in the pelvic cavity. The temperature was 102.4° F.; pulse, 53; respirations, 18. The mucous membranes were slightly congested. The blood picture was as follows:

Total red blood cell count.....10,050,000
Total white blood cell count..... 18,800
Hemoglobin content, 11gm per 100cc of blood, according to the Hadenhauser method.

Differential white cell count (100 cells counted:

Eosinophiles	1	Stab cells	12
Basophiles	1	Segmenters	30
Myelocytes	5	Lymphocytes	41*
Juveniles	5	Monocytes	5

Diagnosis.—Chronic pyometra and adhesions of the genital tract.

Treatment.—An artificial opening through the right side of the vagina into the uterus was made about four inches from the lips of the vulva. A medium-sized stomach tube was inserted into the uterus and a large amount of cream-colored pus was slowly siphoned out. Five cubic centimeters of pituitary extract was given subcutaneously to involute the uterus. Immediately following the removal of the material from the uterus the animal showed severe symptoms of shock; colicky pains, throwing herself, rolling, pawing, spasmodic contractions of the extensor muscles of the legs and neck, respirations greatly increased, with gasping at intervals, pulse weak and irregular. Symptomatic treatment for the shock consisted of intravenous injection of 1,000cc of 25% dextrose solution, to which 2cc of epinephrine had been added, followed ten minutes later by 1,000cc of physiological saline solution administered intravenously, and 30cc of camphorated oil injected intramuscularly in the pectoral region. A few minutes later the mare was assisted to her feet and exercised slowly for about 20 minutes, then placed in a box stall. One hour later she had completely recovered from the shock.

Aftertreatment consisted of daily injections of four ounces of iodine oil (one dram of iodine crystals in one gallon of mineral oil) into the uterus for the first four days. The artificial opening into the uterus was examined daily and kept open.

On October 6, 1939, the mare was discharged from the hospital. The uterus had involuted practically to its normal size, with only a slight discharge.

Laboratory examination showed that the pus from the uterus contained Beta Hemolytic Lancefield C Streptococcus, classed according to Sherman as "Animal Pyogenes."

The material from the uterus, except for about one gallon which escaped before the tube was inserted, was collected in buckets and weighed, totaling 229 pounds. After the drainage the animal weighed 692 pounds; the material in the uterus therefore amounted to about one-third of the animal's normal body weight.

* Twenty-five large, nine atypical, seven small.

Since the animal was suffering no particular pain before, during or after the drainage of the uterus, we can account for the shock only by changes in the circulation incident to relieving the pressure caused by the enormous quantity of pus in the uterus. This permitted a great quantity of blood to suddenly collect in the splanchnic circulation, producing much the effect that would be produced ordinarily by excessive hemorrhage.

P. E. KIMBALL, K. S. C. '40.

Manhattan, Kans.

Chronic Arsenical Poisoning

Chronic arsenical poisoning is liable to occur wherever extensive and heavy and sometimes negligent spraying operations of arsenic have been conducted during the locust campaigns. The veld and water reservoirs are poisoned, although the toxicity diminishes with time and in many instances becomes negligible. There are unfortunately, however, still farms on which no stock can thrive and for years to come will be dangerous to any animal kept long enough to allow of the slow but certainly deleterious effects of chronic arsenical poisoning to develop. Farms are known where up to 95% of the cattle showed the clinical symptoms of chronic arsenical poisoning, the majority of them having enlarged joints.

Steyn (1934) has observed that in cases of chronic arsenical poisoning in cattle enlargement of the joints is a symptom practically always seen. Even in young calves exposed to poisoning for periods of three to six months he has noticed prominently enlarged joints. The enlargements are much more prominent in the forelegs, but are not necessarily bilateral. Adult animals may show tremendously enlarged knees, which are hard and painful; lameness and stiffness supervene (osteitis). Synovial structures are not usually affected, but the articular cartilage may be ulcerated (arthritis). The enlargement is due to thickening of the ends of the long bones coupled with the development of thick fibrous tissue around the joint (periarthritis). Poor condition, according to the degree of poisoning, is usually seen in affected animals, and some may die from unthriftiness.—*J. G. van der Wath,* in the *Jnl. of the South African Vet. Med. Assn.* 10:3.

The Discovery of Vitamin D

About 20 years ago, studies made by the British investigator, Edward Mellanby, indicated that rickets in puppies resulted from a deficiency of some unknown factor, of which cod-liver oil appeared to be a good source. This unknown rickets-preventing factor was erroneously assumed to be vitamin A, since the existence of vitamin A in cod-liver oil had already been established. In 1922, McCollum *et al.,* at Johns Hopkins, conducted an experiment in which, by oxidizing cod-liver oil, they destroyed its vitamin A potency without destroying its antirachitic properties. From this it was established that the rickets-preventing factor is distinct from vitamin A and the newly discovered factor was named vitamin D.

Vitamin D was found to be present not only in cod-liver oil but also in the liver oils of other fish, such as the tuna and sardine. It was also found, in much lower concentrations, in egg yolk and butter fat. Hence, it was classed as a fat-soluble vitamin. Further research showed that it occurred in the non-saponifiable portions of these fats, that is, the sterol fraction, and was apparently a sterol itself. Now it is known that, in fish liver oils, there are two or more sterols having the properties of vitamin D, and that they exist in varying proportions. It is also known that, through activation with ultraviolet irradiation from the sun or from special lamps, vitamin D can be formed from certain sterols (or provitamins) in the skin of animals and in some foods. The multiple nature of vitamin D has been disclosed by studies with chicks, which react differently to different forms. However, many studies have shown that mammals respond equally to different forms of vitamin D. In other words, tests on rats reliably indicate the effectiveness of different sources of vitamin D for four-footed animals.—*Vitamin D Digest.*

Efficacy of Recrystallized Phenothiazine Nematodes from the Gastrointestinal Tract

Introduction

In an earlier paper (Harwood, Habermann, and Jerstad[1]) it was shown that commercial phenothiazine[2] is a highly effective medicament for the removal of nematodes from sheep. Because of the relatively large size of the therapeutic dose, the administration of the drug proved somewhat difficult. It was recognized that commercial phenothiazine was an impure product, and, accordingly, a study of the recrystallized chemical suggested itself as a possible solution to the difficulty mentioned. The data obtained with the recrystallized product are presented in this paper.

Historical

Phenothiazine has been employed as an insecticide for a number of years (Smith[3]) and the toxicology and biochemistry of this chemical has been investigated by Eddy, Cox, and DeEds,[4] Thomas, McNaught, and DeEds,[5] and DeEds, Eddy, and Thomas.[6] Harwood, Jerstad and Swanson[7] reported the use of phenothiazine for the removal of nodular worms from swine and later Harwood, Habermann, and Jerstad[1] found that commercial phenothiazine in large doses was efficient for the removal of round worms from sheep. McNaught, Beard, and DeEds[8] reported that there were 70% fewer encysted trichinæ in rats which had been fed phenothiazine than in control rats which had been similarly infested but kept on a normal diet.

While preparing this manuscript the attention of the writers was directed to a valuable paper by Swales[9] who reported on a practical method of administering phenothiazine to sheep, and stated, "Phenothiazine . . . is here considered to be of exceptional value for the control of parasitic diseases of sheep."

Materials and Methods

Crude phenothiazine was obtained from commercial sources and was recrystallized for the writers by Mr. Harry D. Anspon, at the University of Maryland, by dissolving the commercial product in toluene.

The cull rams and ewes used in this study, which was conducted at the Agricultural Research Center, were purchased in areas close to Beltsville, Maryland. Sheep Nos. 7, 8, and 9 were one year old; all other sheep were aged animals. The species of worms present in each animal were determined prior to treatment by the examination of the feces for nematode eggs. The eggs were identified for the writers by Dr. Doys A. Shorb, of the Zoölogical Division.

The drug was administered either in a capsule or mixed in the feed without preliminary fast and without purgation of any type. When given in the feed, recrystallized phenothiazine was first mixed thoroughly with an equal quantity of molasses and then placed in a mortar with three times as much alfalfa leaf meal and a few pinches of salt. This mixture was ground to a fine pulp and placed before the sheep, which consumed the mixture on the same day that it was offered. After treatment, all the sheep were placed in small, clean pens having concrete floors, and fed a ration of bran and oats. By means of the usual tech-

[1] Harwood, Paul D., Robert T. Habermann, and Arthur C. Jerstad, 1939. Efficacy of Commercial Phenothiazine in the Removal of Roundworms from Sheep. *Vet. Med.* 34, pp. 440-443.

[2] An application for a patent covering the use of phenothiazine as an anthelmintic has been filed.

[3] Smith, L. E., 1937. The Use of Phenothiazine as an Insecticide. U. S. Bureau of Entomol. and Pl. Quar. Cir. E-399.

[4] Eddy, E. W., Alvin J. Cox, and Floyd DeEds, 1937. Studies on Phenothiazine. II. Continued Feeding of Phenothiazine. *J. Indust. Hyg. and Toxicol.* 19, p. 574.

[5] Thomas, John O., James B. McNaught, and Floyd DeEds, 1938. Studies on Phenothiazine. VI. General Toxicity and Blood Changes. *J. Indust. Hyg. and Toxicol.* 20, pp. 419-427.

[6] DeEds, Floyd, C. W. Eddy, and John O. Thomas, 1938. Studies on Phenothiazine V. Fate of Phenothiazine in the Body. *J. Pharm. and Exper. Therap.* 64, pp. 250-262.

[7] Harwood, Paul D., Arthur C. Jerstad and Leonard E. Swanson, 1938. The Efficacy of Phenothiazine for the Removal of Ascarids and Nodular Worms from Swine. *J. Parasitol.* 24, suppl. pp. 16-17.

[8] McNaught, James B., Rodney R. Beard, and Floyd DeEds, 1939. Effects of Sulfanilamide, Phenothiazine, and Thionol in Experimental Trichinosis. *Proc. Soc. Exper. Biol. and Med.* 41, pp. 17-20.

[9] Swales, W. E., 1939. Tests of Phenothiazine, a Highly Efficient Anthelmintic. *Canad. J. Comp. Med.* 3, pp. 188-194.

for the Removal of of Sheep

By ROBERT T. HABERMANN and PAUL D. HARWOOD, Washington, D. C.
Zoölogical Division, U. S. Bureau of Animal Industry

nique, the feces from each treated animal were collected daily and examined for worms. This procedure was followed for the large worms that could be picked out easily without the aid of a dissecting microscope; but for the smaller worms, five or 10 samples, each weighing two grams, were taken from the feces after the total quantity eliminated over a period of 24 hours had been thoroughly mixed and weighed. Each sample was washed and examined separately under a dissecting microscope. As this technique was time-consuming, it was possible to apply it to only a few of the total number of animals involved in the investigation.

As soon as a sheep ceased eliminating worms, usually on the seventh day after the administration of the drug, it was killed and the entire gastrointestinal tract was examined carefully for all nematodes still present. For the large worms, such as Hæmonchus, Ostertagia, Bunostomum, Oesophagostomum, Chabertia, Nematodirus and Trichuris, the usual technique was followed. For estimating the number of small worms, such as Cooperia, Trichostrongylus, Strongyloides and Capillaria, only one-tenth of the total quantity of ingesta and scrapings from the wall of the small intestines was examined under a dissecting microscope.

Details of the results obtained by the experimental administration of recrystallized phenothiazine are presented in Table I.

As cull animals were used in these experiments, it happened that several individuals were very close to death when obtained for experimental treatment. Sheep No. 1 refused all food prior to treatment and died 24 hours thereafter. Sheep No. 2 suffered from a prolapse of the uterus at the time of treatment, and at necropsy exhibited an extensive nephritis. Sheep No. 3 refused all

TABLE I.—The Efficacy of Recrystallized Phenothiazine for the Removal of Strongyloidea from Sheep

Host designation and sex	Weight (Pounds)	Treatment: Dose (Grams)	Treatment: Method of administration[1]	Treatment: Date (1939)	Parasites removed: Bunostomum	Parasites removed: Oesophagostomum	Parasites removed: Chabertia	Parasites removed: Not identified	Necropsy: Date (1939)	Necropsy: Worms found: Bunostomum	Necropsy: Worms found: Oesophagostomum	Necropsy: Worms found: Chabertia
1 Female	56	14.	C	Jan. 14	1	3	0	6	Jan. 16	0	0	0
2 Female	123	24.6	C	Apr. 10	2	1	0	0	Apr. 21	53	4	0
3 Female	61	15.25	C	Jan. 16	7	35	30	6	Jan. 23	0	91	29
4 Female	100	20.	L	Mar. 28	0	6	0	1	Apr. 4	3	0	9
5 Female	97	9.7	F	Jan. 24	0	6	1	0	Jan. 31	43	22	2
6 Female	98	9.8	F	Mar. 7	0	9	10	0	—[2]	38	437	37
7 Male	63	10.	C	May 23	2	40	0	0	June 9	3	55	0
8 Male	101	10.	C	May 23	1	15	0	0	June 9	24	2	0
9 Female	78	10.	C	May 23	4	3	0	0	June 9	17	0	0
10 Female	60.5	12.1	F	Mar. 11	0	41	5	8	Mar. 18	196	20	5
11 Male	156	15.6	F	Jan. 26	3	26	3	2	Feb. 2	33	3	0
12 Febale	84	16.8	C	Mar. 20	15	0	5	2	Mar. 27	60	1	0
13 Female	86	17.2	C	Mar. 31	25	2	0	5	Apr. 7	7	12	0
14 Female	76	19.	F	Jan. 13	12	25	0	8	Jan. 20	0	0	0
15 Female	98	19.6	F	Mar. 14	18	417	14	9	Mar. 22	20	20	23
16 Female	131	20.	C	Apr. 17	6	0	0	0	Apr. 27	39	0	0
17 Female	96	20.	C	Apr. 26	3	39	0	11	May 4	137	16	2
18 Female	93	20.	C	May 12	0	121	1	6	May 19	6	12	0
19 Female	110	22.	C	Mar. 21	16	6	0	2	Mar. 28	3	0	2
20 Male	96	24.	F	Jan. 31	115	43	6	3	Feb. 7	8	1	0
21 Female	120	25.	C	June 13	0	5	0	0	June 23	3	5	0
22 Female	85	25.	C	June 22	47	12	0	12	July 5	3	7	0
23 Male	105	26.25	F	Jan. 20	42	0	24	2	Jan. 27	4	0	0
24 Male	127	31.75	F	Feb. 1	207	2	0	9	Feb. 8	1	0	0

[1] C The phenothiazine was administered in capsules .
F The phenothiazine was administered mixed in feed.
L The phenothiazine was administered by placing loose powder on back part of tongue.

[2] Since sheep No. 6 passed very few nematodes following treatment with 9.8 grams of phenothiazine, it was not necropsied, but redosed on March 14 with 19.6 grams of the drug. This experiment is reported in the above table as sheep No. 15.

food prior to treatment, and at necropsy exhibited a large abscess attached to the cecum and an extensive typhlitis. Sheep No. 4 exhibited a diminished appetite prior to treatment, and at necropsy, a nail one and one-half inches long, surrounded by

TABLE II.—The Average Efficacy of 15.6 Grams to 20 Grams per Dose of Recrystallized Phenothiazine for the Removal of Nematodes from Sheep.

Parasite	Sheep Infested	Worms Present	Worms removed by treatment	Worms found at necropsy	Sheep positive at necropsy	Efficacy of drug (Per cent)
Bunostomum trigonocephalum	8	384	82	302	8	21.4
Oesophagostomum columbianum	7	694	630	64	6	90.1
Chabertia ovina	5	48	23	25	2	47.9
Hæmonchus contortus	4	not determinable	66	0	0	100
Ostertagia spp.	5	not determinable	1	152	4	Greater than 20.0

extensive lesions, was found in the reticulum.

Treatment was administered to these sheep although it was doubtful if peristaltic action was sufficiently vigorous in some of these animals to carry the drug to the worms located in the lower portions of the gastro-intestinal tract. Therefore, for the nematodes located in that part of the intestine, namely, Bunostomum, Chabertia and Oesophagostomum, Sheep Nos. 1-4 do not constitute valid experimental subjects. However, recrystallized phenothiazine was very effective in destroying the worms of the upper part of the digestive tract, since in the three sheep of this group that were parasitized with Hæmonchus, the drug was 100% effective; in the three sheep infested with Ostertagia, the drug was 100% effective in two ewes; but in the third ewe 45 specimens of Ostertagia were found at necropsy.

Sheep Nos. 5 to 10, inclusive, were treated with a total dose varying from 9.7gm to 12.1gm per animal, but these doses were ineffective for the removal of the worms from the intestinal tract, as only 2.1% of the Bunostomum, 7.5% of the Oesophagostomum, and 26.7% of the Chabertia, were removed. A total of 1,144 Ostertagia were recovered at necropsy from these six animals. Since all were still infested at necropsy, it is apparent that the doses used were too small to be effective for the destruction of the lesser stomach worms. Three of the animals from the group were infested with Hæmonchus, against which the medicament was 100% effective. Therefore, it is probable that 10 to 12gm of recrystallized phenothiazine per sheep is an adequate dose for the destruction of Hæmonchus.

Sheep Nos. 11 to 18 were given from 15.6 to 20gm of phenothiazine per dose. Sheep

TABLE III.—The Average Efficacy of 22 Grams to 31.75 Grams per Dose of Recrystallized Phenothiazine for the Removal of Nematodes from Sheep.

Parasite	Sheep Infested	Worms Present	Worms removed by treatment	Worms found at necropsy	Sheep positive at necropsy	Efficacy of drug (Per cent)
Bunostomum trigonocephalum	6	449	427	22	6	95.1
Oesophagostomum columbianum	5	81	68	13	3	83.9
Chabertia ovina	3	32	30	2	1	93.8
Hæmonchus contortus	5	not determinable	2	1 (immature)	1	80.
Ostertagia spp.	5	not determinable	0	60	3	40.

No. 14 was in very poor condition at the time of treatment, but it appeared to be as healthy as the majority of animals available for use in these experiments. However, six days after treatment this sheep died from causes which could not be determined, but which apparently were unrelated to the medication with phenothiazine. As the drug was administered mixed with the feed and was readily consumed by the ewe which, at the time of treatment, did not show any sign of impending death, it is included among the sound experimental animals. The results obtained from sheep Nos. 11 to 18 are summarized in Table II. These data indicate that the doses used were very effective for the removal of Hæmonchus and Oesophagostomum, partially effective for the removal of Chabertia, but only slightly effective for the removal of Bunostomum.

The number of Ostertagia removed by the drug could not be determined accurately because of the digestion of these nematodes in the intestines, but five of the eight sheep to which 15.6 to 20 gm of phenothiazine were administered, were shown by study of the eggs found in the feces to be infested with

Ostertagia at the time of treatment. Since Ostertagia were not present at necropsy in one of these five sheep, it may be assumed that the drug was at least 20% effective for the removal of this parasite. Since only one sheep, but these worms were not considered in calculating the efficacy of the drug.

Eight Trichuris were present in five animals, as follows: 1, 1, 2, 1, and 3 *Trichuris ovis* were present in sheep Nos. 10, 15, 18,

TABLE IV.—The Efficacy of Recrystallized Phenothiazine for the Removal of Cooperia and Trichostrongylus.

Host Designation and sex	Weight (Pounds)	Dose (Grams)	Treatment Parasites removed			Date (1939)	Necropsy Parasites found	
			Cooperia	Trichostrongylus	Not identified		Cooperia	Trichostrongylus
16 Female	131	20	0	13	0	Apr. 27	540	0
17 Female	96	20	0	91	0	May 4	1590	1180
18 Female	93	20	289	1457	195	May 19	1190	1810
2 Female	123	24.6	0	0	0	Apr. 21	170	60
21 Female	120	25	0	0	0	June 23	180	0
22 Female	85	25	2510	288	936	July 5	1590	50

of the seven sheep known to have been infested with Oesophagostomum at the time of treatment was negative at necropsy, these data suggest that phenothiazine may have removed relatively as many Ostertagia as Oesophagostomum, or 90% of the total present.

Table III summarizes the data obtained from sheep Nos. 19 to 24, which received from 22gm to 31.75gm of recrystallized phenothiazine per sheep. These data indicate that more than 20gm of the purified phenothiazine is necessary to be effective against Bunostomum and Chabertia. Again the available data suggest that recrystallized phenothiazine is as effective for the removal of Ostertagia as for the removal of Oesophagostomum.

The data obtained on the effectiveness of recrystallized phenothiazine for the removal of Cooperia and Trichostrongylus from sheep are presented in Table IV. These data suggest that a dose of 20gm of phenothiazine was only slightly effective for the removal of Cooperia from sheep, but a dose of 25gm of the drug was partially effective. Likewise, a dose of 20gm was only partially effective for the removal of Trichostrongylus from sheep, but a dose of 25gm removed 85.2% of 338 Trichostrongylus from one sheep. It should be noted that numbers of small, unidentifiable nematodes were also removed from these

20, and 24, respectively. Following treatment, 1 and 3 Trichuris were eliminated in the feces of sheep Nos. 18 and 24, respectively. Of 318 Nematodirus found in five sheep, only three were recovered from the feces. No Strongyloides were eliminated, but 170 of these parasites were found at necropsy in one sheep. Twenty *Capillaria brevipes* were obtained from the intestines of one sheep at necropsy, although none were eliminated with the feces. Tapeworms were recovered from sheep Nos. 10, 14, 19, 21, 31, and 32 at necropsy.

Hematocrit readings and temperature records on three sheep were taken to determine if the administration of phenothiazine to these hosts produced any noticeable illness. It was found that the body temperatures were not altered following treatment, and that the volume of erythrocytes was not affected except in sheep No. 22, from which a considerable number of worms was removed. In this case the volume of erythrocytes increased from 19 to 26.5% in 15 days. Apparently, single large doses of phenothiazine do not regularly produce anemia in sheep similar to the anemia observed by Thomas *et al.*[5] in rabbits after an excessively large dose of this drug.

In an attempt to produce symptoms of toxicity, a mature Dorset female was dosed with a total of 247gm of recrystallized phenothiazine during a period of 21 days.

On March 27, 1939, a mixture containing 107gm of recrystallized phenothiazine, 150gm of alfalfa leaf meal and 54cc of molasses was placed before the ewe. Since only a few mouthfuls were eaten, oats and bran were added to the mixture on March 28. The ewe then consumed about half the mixture. Very little of the remaining meal was consumed in the next three days; the remainder of the mixture was, therefore, placed in hard gelatine capsules and administered to the sheep on April 1. The sheep appeared normal until April 10, when an additional 60gm of phenothiazine was administered in four hard gelatin capsules, one of which broke and lodged in the animal's throat. Labored respirations and a discharge of a large quantity of frothy mucus from the nostrils indicated that some of the powder was inhaled. In the afternoon the ewe appeared to have recovered and 40gm more of phenothiazine was administered. The temperature of the ewe had been 102.4°F., but on April 11, the temperature rose to 102.9°F., and the sheep was coughing. On April 12, 40gm of phenothiazine was administered in capsules. At this time the sheep exhibited a mucus discharge from the nose, labored respirations, rales, and a temperature of 104.8°F. The sheep's condition gradually became worse and the temperature gradually dropped until the animal died on the night of April 17. The necropsy findings revealed a well-developed case of broncho-pneumonia, with extension of the infection to the other vital organs.

Since the ewe appeared normal after consuming 107gm of recrystallized phenothiazine, which is about four times the therapeutic dose, and did not show any symptoms until the drug was aspirated into the lungs, it is evident that phenothiazine has a wide margin of safety when administered in therapeutic doses.

Discussion

It is well known that the anthelmintic activity of a substance is usually correlated with its water solubility. The effective range of water solubility for the halogenated hydrocarbons was found to vary from one part of the chemical in 1,000 parts of water, to one part in 5,000 parts of water by Wright and Shaffer,[10] and Wright, Shaffer, Bozicevich and Underwood.[11] Similarly, Lamson *et al.*,[12] found that the effective alkyl phenols were sparingly soluble in water. According to the data presented by Gersdoff and Claborn[13] and by Fink, Smith, Vivian, and Claborn,[14] phenothiazine is extremely insoluble since from 100,000 to 1,000,000 parts of water are required to dissolve one part of the drug. Accordingly, it is doubtful if the worms are destroyed directly by the chemical, phenothiazine. It is well known that this chemical is readily oxidized, and DeEds *et al.*[6] have demonstrated that within the body of rats a portion of the phenothiazine administered by mouth is converted into more soluble substances such as thionol and various conjugation products. As the urine, and to some extent the feces of sheep that have been dosed with phenothiazine are colored red, it is probable that similar chemical reactions take place in the alimentary tracts of these animals, and the effective anthelmintic is probably one or more of these more soluble derivatives which have been identified in part by DeEds *et al.*[6]

The quantity of phenothiazine which reacts chemically in an animal's body is not known, but since Knipling[15] found that the feces eliminated by a cow receiving relatively small quantities of phenothiazine were unfavorable for the development of horn fly larvæ, even after dilution with several times as great a quantity of feces from

[10] Wright, Willard H., and J. M. Schaffer, 1932. Critical Anthelmintic Tests of Chlorinated Alkyl Hydrocarbons and a Correlation between the Anthelmintic Efficacy, Chemical Structure and Physical Properties. *Am. J. Hyg.*, 16, pp. 325-428.

[11] Wright, Willard H., J. M. Schaffer, J. Bozicevich, and P. C. Underwood, 1937. Critical Anthelmintic Tests of Some Primary Monobrom Hydrocarbons. *Rabot. Gel'mint.* (Skrjabin) pp. 769-780.

[12] Lamson, Paul D., Harold W. Brown, Roger W. Staughton, Paul D. Harwood, Richard Baltzly, and Allan D. Bass, 1935. Anthelmintic Studies on Alkyl-hydroxybenzenes. V. Phenols with Other than Normal Alkyl Side Chains. *J. Pharmacol. and Exper. Therap.* 53, pp. 239-249.

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[14] Fink, D. E., L. E. Smith, D. L. Vivian, and H. V. Claborn, 1938. Toxicity Tests with Synthetic Organic Compounds against Culicine Mosquito Larvæ. U. S. Bureau of Entomol. and Pl. Quar. Circ. E-425.

[15] Knipling, E. F., 1938. Internal Treatment of Animals with Phenothiazine to Prevent Development of Hornfly Larvæ in the Manure. *J. Econom. Entom.* 31, pp. 315-316.

a normal animal, it is probable that a large part of the phenothiazine administered to a ruminant is voided in the feces. Phenothiazine is the only drug known at the present time which is effective when administered by mouth in a single dose for the removal of *Oesophagostomum columbianum*, and it is possible that this action is due to the extreme insolubility of the compound in connection with its slow rate of change to more soluble and effective anthelmintics.

On the basis of experiments reported in this paper, and of the extensive researches of DeEds and his co-workers, it is believed that phenothiazine is a relatively safe anthelmintic for administration to sheep. Probably the insolubility of phenothiazine precludes rapid absorption of this chemical, and the reaction products which are produced slowly are readily eliminated by the kidneys following absorption. While it is impossible at this time to designate the therapeutic index of phenothiazine as an anthelmintic for sheep, there exists sufficient evidence to indicate that this figure is greater than one to four.

In an earlier paper, Harwood, Habermann, and Jerstad[1] reported that commercial phenothiazine was an effective drug for the removal of nematodes from sheep, but that the drug was too bulky to be administered under practical conditions. Recrystallization has greatly reduced the quantity necessary for effective medication of sheep, and rendered its administration a practical measure. The increased effectiveness of recrystallized phenothiazine is not due wholly to the removal of inactive impurities, since better than 90% by weight of the commercial product was recovered in the recrystallized phenothiazine. A dose of 20gm may be given in a single hard gelatine capsule having a capacity of 1½ ounces. However, for the removal of Bunostomum and Trichostrongylus spp., a larger dose (25gm) is desirable and it is necessary to administer two one-ounce capsules when this dose is given.

Furthermore, sheep eat food mixtures containing recrystallized phenothiazine more readily than food mixtures containing the less pure product. It is possible that flock treatments with recrystallized phenothiazine administered in the food will prove feasible in areas where supplementary feeding of concentrates is practiced, but additional data will be required before this method of administration can be recommended.

The data presented in this paper show that recrystallized phenothiazine in doses of 20gm for sheep weighing more than 70 pounds proved very effective for the removal of nodular worms, *Oesophagostomum columbianum*, common stomach worms, *Haemonchus contortus*, and lesser stomach worms, Ostertagia spp. In doses of 25gm it also exhibited a high efficacy for the removal of the hookworm, *Bunostomum trigonocephalum*, the large-mouthed bowel worm, *Chabertia ovina*, and apparently was fairly effective for the removal of the small trichostrongyles, Trichostrongylus spp., and Cooperia spp.

Using a different approach to the problem, Swales[9] has developed a bolus, consisting of phenothiazine, which disintegrates rapidly in the gastrointestinal tract of sheep. Since this bolus is formed under high pressures (4,000 pounds per square inch) the total mass is greatly reduced, and it may be given to sheep under practical conditions. Apparently, Swales used phenothiazine which had not been recrystallized. Consequently, it is possible that a bolus similar to the one employed by Swales, but made of recrystallized phenothiazine, will prove to be superior to either method of administration developed up to the present time.

Recrystallized phenothiazine is not available commercially at the present time, but there are indications that this chemical will soon be available in form suitable for administration to sheep.

The Significance of Variations of the Magnesium Content of the Serum of Swine

By D. F. EVELETH and T. W. MILLEN, Ames, Iowa*
Veterinary Research Institute, Iowa State College

THE importance of magnesium in mammalian metabolism was shown very clearly in the experiments of Kruse, Orent and McCollum[1]. Since that time much has been published on the study of magnesium metabolism of animals and human beings. These studies have recently been reviewed by Greenberg[2] and more completely by Duckworth[3]. Data on magnesium metabolism in swine are, however, for the most part, lacking or where such data have been published, only incidental to other studies.

The blood or serum magnesium content offers a means of studying magnesium metabolism in intact animals showing no symptoms of magnesium deficiency. The methods used in this laboratory for the determination of serum magnesium have been discussed previously[4].

Studies on the magnesium metabolism in swine were initiated in this laboratory as a result of an observation made in 1933. Swine suffering from severe posterior incoördination were found to have an elevated serum magnesium due to drinking water high in magnesium sulphate. Since that time the serum magnesium determinations have been made on a large number of swine blood samples. We have published reports in which the values of the serum magnesium were given for experimental animals used in certain physiologic and pathologic studies. These data, while few in each case, established certain trends or indications of what the normal serum magnesium of the pig might be. The range encountered has been from 1 to 6.5mg %. The actual variations in milligrams were usually not large but the percentage changes were great. The feeding of many types of rations was tried. The serum magnesium of a group of animals under similar environmental conditions would be similar but in comparing these with other groups under different conditions, large discrepancies were found. Frequently, during the early part of the work, rations were fed which we have later found to be definitely inadequate for proper nutrition in the pigs.

In attempting to correlate the blood serum magnesium with other constituents no correlation could be found between magnesium and chlorides, phosphates, calcium or glucose. There did appear to be a positive correlation with serum magnesium and total serum protein. Greenberg, Lucia and Tufts[5] have found a progressive decline in the serum protein of rats fed rations deficient in the magnesium. In no cases have we encountered the low serum magnesium in swine that have been reported in other species.

Data obtained in some of the feeding experiments illustrate the effects of diet on the serum magnesium of the pig. A few typical examples are given in Table I. These animals were all penned inside. They were given tap water and those not receiving tankage were given an iron and copper supplement. All of these animals, with the exception of the day-old pigs, were given cod-liver oil as a source of vitamins A and D. With the exception of the day-old pigs, they weighed between 40 and 150 pounds at the time the blood samples were taken. Ration No. 1, with added MgO, definitely

* Doctor Eveleth is now at Fayetteville, Ark., as head of the Department of Bacteriology and Veterinary Science, University of Arkansas; Doctor Millen is at Sioux City, Ia.

[1] Kruse, H. D., E. R. Orent and E. V. McCollum, 1932. Studies on Magnesium Deficiency in Animals. I. Symptomatology Resulting from Magnesium Deprivation. *Jour. Biol. Chem.* 96, 519.

[2] Greenberg, D. M., 1939. Mineral Metabolism, Calcium, Magnesium and Phosphorus. *Am. Rev. Biochem.* 8, 269.

[3] Duckworth, J., 1939. Magnesium in Animal Nutrition. *Nutr. Abst. and Rev.* 8, 841.

[4] Eveleth, D. F., 1937. Comparison of the Distribution of Magnesium in Blood Cells and Plasma of Animals. *Jour. Biol. Chem.* 119, 289.

[5] Greenberg D. M., S. P. Lucia and E. V. Tufts, 1938. The Effect of Magnesium Deprivation on Renal Function. *Am. Jour. Physiol.* 121, 424.

increased the serum magnesium of the pig, while ration No. 5, with added $CaCO_3$, pro-

TABLE I.—The Effect of Diet on the Magnesium Concentration of Swine Serum

Ration No.	Constituents		No. Animal	Range (Mg. Per Cent)	Mean
1	Soybean meal	20.0	1	—	4.80
	Corn	74.0			
	Yeast	4.0			
	MgO	2.0			
2	Tankage	9.7	5	3.2–3.5	3.30
	Soybean meal	9.7			
	Alfalfa meal	9.7			
	Corn	67.9			
	Bonemeal	3.0			
3	Soybean meal	100.0	1	—	3.30
4	Tankage	10.0	4	3.2–3.3	3.25
	Soybean Meal	10.0			
	Alfalfa meal	10.0			
	Corn	70.0			
5	Soybean meal	20.0	1	—	3.10
	Corn	72.0			
	Yeast	4.0			
	$CaCO_3$	4.0			
6	Corn	80.0	3	2.5–3.1	2.73
	Skimmilk powd.	20.0			
7	Corn	92.0	1	—	2.70
	Yeast	4.0			
	Bonemeal	3.0			
	NaCl	1.0			
8	Sow's milk	100.0	6	2.3–2.9	2.67
9	Tankage	10.0	3	2.3–2.7	2.53
	Alfalfa meal	10.0			
	Corn	77.0			
	NaCl	1.0			
	Bonemeal	2.0			
10	Polished rice	100.0	1	—	2.50
11	Corn	77.0	3	2.1–2.5	2.33
	Skimmilk	20.0			
	$CaCO_3$	3.0			

duced a slight decrease. Rations Nos. 2, 3 and 4 are all relatively high in magnesium and protein. Ration No. 3 is unsatisfactory because of other dietary deficiencies. Rations Nos. 2 and 4 are apparently nearly optimum as regards magnesium and other factors, as the growth and appearance of the pigs were very good.

Comparison of rations No. 6 and No. 11 shows clearly the effect of adding $CaCO_3$ to a ration low in magnesium. When the magnesium of the ration is high the addition of calcium causes little decrease in the serum magnesium while with rations low in magnesium a definite decrease occurs. Sow's milk appears to be slightly low in magnesium. Two pigs, 10 days old, however, had serum magnesium of 3.1 and 3.2mg % respectively.

The results of these and other analyses indicate that the optimum serum magnesium of the pig is between 2.9 and 3.4mg%. This value is somewhat higher than for other species. Data on serum calcium, potassium and sodium show them all to be slightly higher for swine than for most other species.

Physiologic and pathologic data on the significance of changes in the serum magnesium are in many respects similar to those of inorganic phosphate. The effect of the dietary regime may completely mask

Diseases of swine which impair locomotion appear to be produced by a large number of different etiological factors. Deficiencies or serious imbalances of calcium and phosphorus are known to produce severe incoördination, lameness and deformed bones in swine. Vitamin D deficiency also causes a very severe form of lameness in swine. These preliminary studies suggest that some of the cases of posterior incoördination of swine encountered under field conditions are associated with a hypomagnesemia. However, the significance of this observation cannot be evaluated from the data available.

The interrelationship of magnesium metabolism with that of calcium and phosphorus has been shown in studies on cattle. There appears to be slight danger of a magnesium deficiency in most rations fed swine. Many rations fed swine are relatively high in magnesium salts. The investigations reported here do not indicate that moderately high magnesium rations exert a deleterious effect on swine. These data do indicate that the magnesium content of swine serum is influenced by the magnesium, calcium and phosphorus content of the ration.

These studies show that when both the calcium and phosphorus content of the ration are adequate and there is sufficient vitamin D available there is no disturbance in the magnesium content of the serum.

the effect produced by either a physiologic or pathologic disturbance.

Serious magnesium deficiency in swine as a result of inadequate amounts of magnesium in the rations appears unlikely. Pigs fed insufficient protein grow slowly so that unless protein is added there is little likelihood of magnesium deficiency. When a protein supplement is added the magnesium intake of the ration will be increased. With the exception of skimmilk powder most protein supplements are relatively high in magnesium.

TABLE II.—The Serum Calcium and Magnesium of Swine Suffering from Various Diseases

	Diagnosis	Calcium (Mg. Per Cent)	Magnesium (Mg. Per Cent)
1.	Cholera with posterior incoördination	9.6	2.0
2.	Posterior incoördination	10.4	2.4
3.	Posterior incoördination	9.5	2.2
4.	Peritonitis	8.3	2.1
5.	Chronic oxalic acid poisoning	10.5	2.3
6.	Vitamin D deficiency	6.1	2.6
7.	Vitamin D deficiency	5.0	2.4
8.	Vitamin D deficiency	6.6	2.6
9.	Posterior incoördination	10.2	4.1
10.	Posterior incoördination	10.0	3.6
11.	Calcium deficiency	8.7	4.1
12.	Calcium deficiency	8.5	3.5
13.	Moribund cholera	11.9	4.2
14.	Moribund CCl_4 poisoning	12.0	6.5

An interrelationship of protein, calcium and magnesium seems indicated. A deficiency in any one probably causes disturbances in the balance of the other two. A few selected data obtained on pathologic blood sera illustrate the calcium to magnesium relationship in some conditions. These are given in Table II. The serum calcium of normal swine is usually between 10.8 and 12.0mg %. In all but two cases in Table I there is a definite subnormal serum calcium. The magnesium falls into three distinct classes: the hypomagnesemia in one type of posterior incoördination, the slight hypomagnesemia in vitamin D deficiency, and the hypermagnesemia in another type of posterior incoördination and in simple calcium deficiency. We have reported low calcium with little change in magnesium in the plasma of swine suffering from hog cholera[6]. Johnson and Palmer[7] have discussed the very low plasma calcium of swine suffering from vitamin D deficiency. In most diseases, when the animals reach a moribund state there is an increase in both calcium and magnesium. This is illustrated in cases Nos. 13 and 14.

In case No. 5 the chronic oxalic acid poisoning in the pig did not produce noticeable symptoms. The ration was composed of corn 72.75%; alfalfa meal 4.85%; tankage 4.85%; skimmilk powder 14.55%; and oxalic acid 3.0%. The animal was slightly nervous but otherwise showed no ill effects. Over a feeding period of 66 days the animal gained from 96 to 170 pounds in weight.

The serum magnesium levels for swine reported by Eveleth and Biester[8] were in many cases definitely higher than those found in this study. There are several possible interpretations of those results. The animals killed after approximately four months on the rations had lower plasma magnesium than those killed later. These values are more in accord with the data in Table I, namely, that low magnesium—high calcium rations tend to depress the serum magnesium. The animals killed later had much higher serum magnesium. This could be in part due to the slow growth of the animals fed the low skimmilk powder rations. There may have been an adjustment on the part of the animals to maintain a normal serum magnesium. In the fourth group, receiving a high protein—high calcium diet there may have been an equilibrium established with a high serum protein and a high serum magnesium.

Summary

1. The serum magnesium of swine fed different rations has been determined. The data indicate that the serum magnesium is influenced by both the magnesium content of the ration and the calcium content of rations low in magnesium.

2. A few data on swine suffering from various diseases are used to illustrate the calcium and magnesium relationship in a few common diseases of swine.

[6] Eveleth, D. F. and L. H. Schwarte, 1939. Chemical Changes in the Blood of Swine Infected with Hog Cholera. *J. A. V. M. A.* 94, 411.

[7] Johnson, D. W. and L. S. Palmer, 1939. Individual and Breed Variations in Pigs on Ration Devoid of Vitamin D. *Jour. Agr. Res.* 58, 929.

[8] Eveleth, D. F. and H. E. Biester, 1938. Blood Chemistry in the Study of Nutritional Diseases of Swine. *J. A. V. M. A.* 93, 249.

Swine Production

According to reports there were approximately 80 million pigs produced in the United States during 1939. This includes both spring and fall litters. Of the 80 million, over 28 million were produced in seven states: Iowa, Illinois, Minnesota, Nebraska, Missouri, South Dakota and Kansas.

About 60% of the 80 million will survive until marketed; the other 40% will die before they reach the marketable age.

One of the major veterinary problems is the prevention of pig losses.

Loss of Swine Following Feeder Cattle

A feeder purchased some 40 shotes from a farmer and placed them in a cattle feed lot with about 60 native shotes. The cattle were fed corn and kaffir corn.

The recently-acquired shotes weighed from 90 to 100 pounds. They had been vaccinated against cholera and were apparently healthy and normal, but about a week after they had been placed in the cattle feed lot some of them became visibly sick. The native shotes remained healthy. The affected animals had a diminished appetite and most of them had diarrhea. In some cases the feces were admixed with blood. The temperature was normal. Death occurred in 24 to 48 hours.

Autopsy revealed relatively extensive enteritis involving the mucous membrane of the stomach, small and large intestine. The mesenteric lymph nodes were extensively congested and hemorrhagic. The spleen was apparently slightly tumefied. A few small spherical bodies, probably protozoa, were found in microscopic smears from the affected mucosa. Cultures from the mucosa, spleen, kidney and heart blood were negative at 24 and 48 hours.

The perplexing problem in this report is the extensive involvement and yet the condition was afebrile and affected only one group of hogs on the premises. So far as known, this malady has not occurred in any other lot of swine in this community.

What is the disease?—*H. L. B.*

Eclampsia in a Sow

I have a client who recently bought a very fine prize-winning sow. This sow farrowed eight pigs during a frightful heat wave; immediately thereafter she showed severe nervous symptoms—walking about, gasping, champing the jaws, and finally collapsing.

I diagnosed the condition as eclampsia and resorted to injections of calcium gluconate. Some improvement was apparent, but mastitis developed. This condition was treated with sulfanilamide combined with sodium bromide. Again some improvement was noted, but the sow had a relapse and died. The temperature was 106° F. the day prior to death. Prior to this the temperature had been normal.

Autopsy revealed a normal condition of all organs except that the spleen was enlarged and the lungs were full of edematous fluid.

Was the condition in this sow primarily eclampsia or was overheat of importance? Comments relative to this case by readers of VETERINARY MEDICINE will be appreciated.—*E. J. F.*, Cal.

Septicemia in Swine

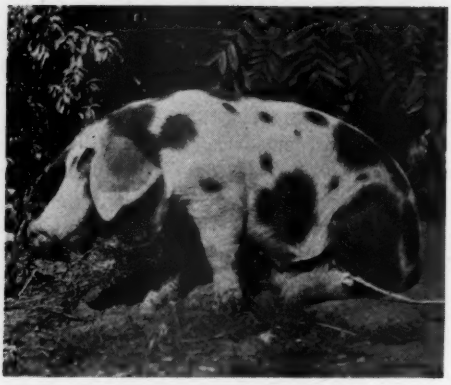
I have a client who from time to time loses one of his large hogs. These losses have occurred over a period of several months. These hogs take sick suddenly and become gaunt rapidly and there is elimination of quantities of dark colored feces.

The affected hogs usually die in four or five days. An occasional case recovers. On autopsy the stomach is relatively full. The small intestine is quite congested. The mesenteric lymph nodes are blue-black in color and tumefied, and there are usually red blotches in the gastric mucosa.

What is this condition?—*W. H. B.*

Comment: A laboratory examination of specimens from one of the hogs yielded cultures of streptococcus that possessed diphtheroid characteristics after 72 hours from all tissues examined.

It is our judgment that the streptococci identified in these specimens were respon-



Streptococcic Septicemia

sible for the occasional cases of septicemia and death. This condition rarely becomes a herd problem.

Further discussion by readers of VETERINARY MEDICINE will be appreciated.—*A. T. K.*

Actinomycosis in Swine

Actinomycosis is relatively prevalent in swine in some sections of the cornbelt. This condition has been observed mostly in slaughtered hogs, although occasional cases have been recognized by practitioners. The usual symptom consists of tumefaction in the pharyngeal region and there may or may not be a swelling in the cervical region.

On post-mortem examination there are usually found numerous purulent necrotic foci about the size of an ordinary pea. The purulent areas are usually encapsulated. The pus is not as granular and stringy as bovine actinomycotic pus.

Actinomyces can usually be demonstrated by laboratory tests. The rays of the fungus are relatively thin and long. Pure cultures of a small streptococcus may be isolated from the lesion; these may be the streptococcus of the so-called swine strangles.

Of the source of the fungus and the method of infection little is known.

The administration of potassium iodide in small doses in the feed over a period of two or three months usually suffices as treatment.

Comments and reports of cases of porcine actinomycosis and methods of treatment are invited.—*A. T. K.*

Corynebacterial Infection In Swine

According to a recent study by Bendixen and Jepsen, the *Corynebacterium equi* may be either acid or non-acid-fast. The authors do not believe that the acid-fast bodies are spores; however, they seem to be more resistant than the non-acid-fast rods and spheres. Again it has been noted that the acid-fast organisms increase in number as the lesion increases in age.

It is also conceded there is a need for a more systematic bacterial classification. Jensen is of the opinion that the *Corynebacterium equi* is a soil bacterium that may infect swine. It has been demonstrated that swine are readily infected experimentally through the nose and throat, and by ingestion.

Lung lesions of varying extent occurred in experimentally infected swine, but there was little tendency for the infection to become generalized. In some experimental animals there were lesions in the cervical lymph nodes, but in only one instance has an infected mesenteric lymph node been observed.

Cultures have been isolated from gastric and cecal content 19 days after nasal infection. In many instances the corynebacteria have been isolated from apparently healthy cervical lymph nodes of inoculated and non-inoculated swine. The lesions produced by the corynebacterium are of an inflammatory character and may be acute or chronic.

The lung lesion reminds one of the lesion of tuberculosis except that the healing by production of connective tissue is very pronounced. In some cases, about four weeks after infection, the lymph nodes show necrotic foci surrounded by a zone of large mononuclear cells and giant cells and later a capsule develops.—*A. T. K.*

Pullorum Disease Control and Eradication*

By HENRY VAN ROEKEL,
Amherst, Massachusetts
Chief of Laboratory,
Department of Veterinary Science,
Massachusetts Agricultural Experiment Station

PULLORUM disease, an infectious, communicable malady, is caused by the bacterium *Salmonella pullorum*. Rettger, who discovered the causative agent in 1899, referred to the disease as bacillary white diarrhea. The present term, pullorum disease, has been in use since 1928.

Pullorum disease is recognized as an economic problem to the poultry industry throughout the world, and as such has been subjected to far-reaching investigations. These have yielded encouraging results, and satisfactory methods for the control, eradication and prevention of the disease have been developed.

Geographical Distribution

The presence of pullorum infection has been reported in Africa, Asia, Australia, Canada, England, continental Europe, South America and every state in the United States. In the United States, the intensity of the infection appears to vary little in degree, with the exception of a few states where concerted efforts have been made toward establishing flocks free of the disease. The general distribution of the disease throughout the country has been brought about in some degree through the advent of modern methods of hatching and distribution of eggs and chicks.

Pullorum Disease Hosts

A wide variety of animals is susceptible to pullorum infection. Among the avian species the bull-finch, canary bird, chicken, duck, goose, guinea-fowl, pheasant, pigeon, sparrow and turkey have been infected with *S. pullorum*. Among the mammalian species the cat, guinea pig, mouse, rabbit and rat were found susceptible. It is quite likely that other species, both avian and mammalian, might be vulnerable to this infection; but under natural conditions, observations have revealed that the disease is quite generally established in the domestic chicken and, to some degree, in turkeys in certain sections of the country.

Symptoms and Lesions

In the adult birds, the disease usually manifests itself in a chronic form. The organism may localize in various parts of the body but is most frequently found in the ovary. Infected birds may eliminate the organism in the egg, as illustrated in Figure 1. Egg production is affected to a greater or lesser extent in an infected flock. Fertility and hatchability are also reduced. Acute outbreaks may occur among flocks of adult birds. In such instances, the disease follows a definite course, accompanied by a severe mortality. Chronic local infections in birds may develop into acute infections, especially when other debilitating factors are introduced. Infected birds as a rule cannot be differentiated from the non-infected by

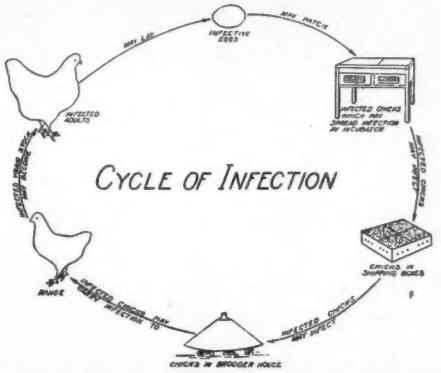


Fig. 1. Pullorum disease infection in a flock may follow this cycle. The disease can be eradicated either by breaking the cycle of infection through eliminating the infected birds by means of the agglutination test, or by disposing of the entire infected flock and replacing it with pullorum disease-free stock.

*Contribution No. 354 of the Massachusetts Agricultural Experiment Station.

physical examination; accurate diagnosis must be based on serological and bacteriological examinations.

In young chicks the disease may manifest itself immediately upon hatching if the infection be severe. Affected chicks may exhibit a tendency to remain under the hover, depressed vigor, loss of appetite, frequent cheeping, droopy wings, shortened bodies, pasting of the vent with white, chalky excreta, and somnolence which may be followed by death. These symptoms are neither characteristic nor specific for pullorum disease alone since they may be observed in other chick diseases. The mortality rate may vary from a so-called "normal" mortality (1-3%) to as high as 100%. Losses may occur immediately after hatching and continue for a period of three or more weeks, with the greatest mortality usually occurring during the second week of chickhood (Figure 2). The onset and severity of the disease are influenced by the amount of infection in the body, the virulency of the organism, the susceptibility of the host, and the care given by the poultryman. In some cases infected chicks exhibit no external signs of the disease, while in others all the possible manifestations of the disease are observed. Lesions in chicks affected by pullorum disease do not furnish definite pathognomonic criteria. An accurate diagnosis depends on the isolation and identification of the causative agent.

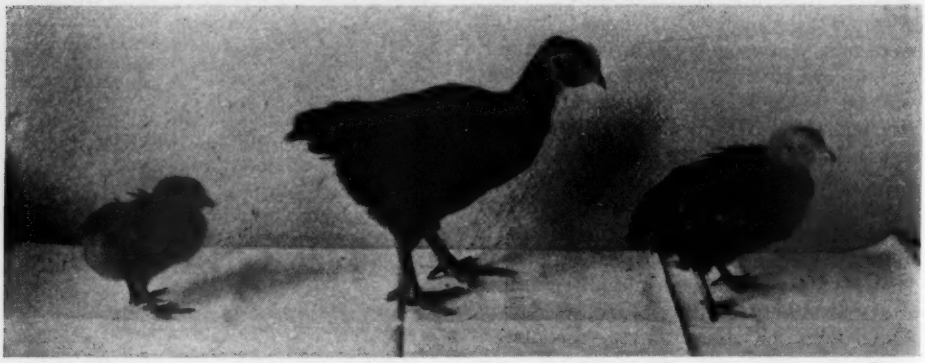


Fig. 2. Six-weeks-old chicks exposed to pullorum infection when 72 hours old. Weights: No. 1, 115gm; No. 2, 488 gm; No. 3, 193gm. Pullorum disease has an influence on the rate, uniformity, and amount of growth.

Transmission of Diseases

The modes of transmission of the disease must be fully appreciated before one can intelligently and effectively apply measures for its control, eradication and prevention. The cycle of infection (Figure 1) has been established for approximately three decades. The dissemination of infection for the most part follows this cycle. However, one should bear in mind that stock may become infected whenever it comes in contact, directly or indirectly, with the source of infection. The causative agent may be eliminated by means of the droppings (excretions from the respiratory, digestive, urinary and reproductive tracts), by eggs and from abrasions in the skin. In adult flocks the greatest spread appears to occur through the eating of freshly laid eggs that are infective. Cannibalism and insanitary environment also increase the infection in a flock. Male birds may contract the disease, and therefore should be considered as possible disseminators of the infection. Male reproductive organs, as well as other organs, have been found infected (Figure 3). Controlled experiments have failed to show conclusively that the male may spread the disease in a flock either through body discharges or as a mechanical carrier in transmitting the organism from the cloaca of an infected female to the cloaca of a non-infected female. However, a more exhaustive investigation of this phase of the

disease is necessary before one can conclude that the infected male is of little concern in the combat against pullorum disease.

Pullorum disease in chicks usually has its origin in the incubator as a result of hatching infective eggs and permitting the infection to spread to non-infected stock. Occasionally it is observed that chicks may contract the disease from incubators contaminated with *S. pullorum*. The disease may continue its spread among chicks when kept in chick boxes, retaining batteries, brooders, etc. Equipment, feed and water contaminated with *S. pullorum* all serve as possible sources of infection. Persons handling infected stock or contaminated equipment and materials may serve as mechanical carriers of the infection.

Control, Eradication and Prevention

Losses from the disease may be mitigated through control measures. The application of the agglutination test, removal of reactors, and improvement of sanitary methods tend to reduce the losses among maturing and adult birds as well as among their progeny. Incubator hygiene and fumigation reduce the spread of the infection. Proper brooder sanitation and management may prevent precipitation of acute outbreaks and serious losses among chicks. However, the most excellent control measures seldom if ever permit assurance against losses from the disease, and the objective in combating the disease is complete elimination of the disease-producing agent.

In 1914 the macroscopic agglutination test was applied for the detection of adult carriers of the infection. It was realized at this time that the only real basis for improvement among breeding flocks was to detect and to eliminate all infected birds. With this fundamental objective in mind, great progress has been made in combating pullorum disease.

Success in eliminating the disease from flocks can be attained through the adoption of an effective eradication program, of which the macroscopic agglutination test represents a part. The effectiveness of the test is only as great as the efficiency of the methods employed and the competency of the person conducting it. The Conference of Laboratory Workers in Pullorum Disease

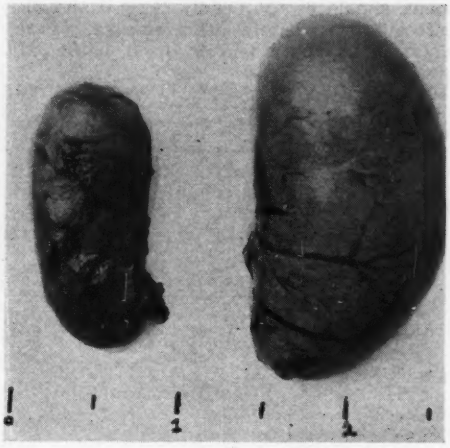


Fig. 3. Testicles removed from a reacting adult bird. Testicle No. I appeared to be functioning even though its structure was firm and its size greatly reduced. *S. pullorum* was isolated from this testicle. Testicle No. II appeared to be normal in size and texture. *S. pullorum* was not isolated from this organ.

Eradication has improved the methods and standardized the technique employed in pullorum disease testing, and has brought about a greater uniformity of interpretation of results. Aside from the agglutination test, other fundamental disease-control and eradication measures are regarded essential for an effective program. One should be cognizant of the fact that pullorum disease is infectious and communicable, and that it may be disseminated as long as infected individuals or infective objects are permitted to have direct or indirect contact with susceptible stock.

The following measures have been found effective.

1. All the birds on the premises should be tested each year.
2. If infection is present, the entire flock should be retested within four to six weeks until a negative report is obtained, provided the value of the birds justifies the expenditure.
3. Every reactor, regardless of its value, should be removed from the premises and sold for slaughter immediately.
4. Offal from all birds dressed for market

or for home consumption, as well as dead birds that are not fit for consumption, should be burned.

5. The poultry houses, runs, and equipment, should be thoroughly cleaned and disinfected immediately after removal of reactors. An empty pen in each house will facilitate cleaning and disinfection during the winter months. Disinfectants approved by the United States Department of Agriculture are recommended.

6. Birds removed from the premises to egg-laying contests, exhibitions, etc., should be held in quarantine and determined to be free of disease before they are readmitted into the flock.

7. Purchase of stock, whether adults, chicks or eggs, should be from known pullorum disease-free flocks. An official testing agency should be consulted regarding additions or replacements.

8. Eggs should not be saved for hatching until after a flock has been tested and all the infected birds removed. Early pullet testing will permit early hatching.

9. Fresh and infertile eggs from unknown or infected sources should not be fed to chickens or exposed to animals such as crows, sparrows and skunks that may carry or spread the infection.

10. Poultrymen should not custom-hatch for untested or infected flocks (including fowl other than chickens).

11. Owners of pullorum disease-free flocks should not have hatching done where infected eggs or stock may be found.

12. Poultrymen should not buy feed in bags that have been used or exposed to infection. (Such bags if properly disinfected will be safe for further use.)

13. Poultrymen should regard fowl other than chickens as a possible source of pullorum infection unless tested and found free from pullorum disease.

14. Poultrymen should not use equipment that has been exposed to or contaminated with infective material unless it is properly cleaned and sterilized or disinfected.

TABLE I. Massachusetts Testing Summary Covering an 18-Year Period

Classification	1920-21	1926-27	1932-33	1937-38
Total flocks tested	108	249	335	308
Total birds tested	24,718	127,327	296,093	480,227
Total tests	24,718	127,327	300,714	497,769
Positive tests (per cent)	12.50	4.03	0.47	0.17
Percentage of total tested flocks non-reacting	23.15	45.78	82.39	92.85
Percentage of total tested birds in non-reacting flocks	9.77	31.63	80.41	95.26

TABLE I-A. Consecutive Years 100% Tested With No Reactors

Consecutive Years	2	3	4	5	6	7	8	9	10	11
Number of Flocks	35	38	20	26	18	2	4	2	5	2

Progress in Eradication

The establishment and maintenance of pullorum-clean flocks is progressing rapidly in some states in this country. Table I gives a testing summary extending over an 18-year period for Massachusetts flocks. The increases in the number of tested flocks, number of tested birds and number of tests, as well as the reduction in the percentage of positive tests, are indeed very encouraging. More striking are the increases during this period in the percentage of total tested non-reacting flocks and percentage of total tested birds in non-reacting flocks. Table I-A also points out that some flocks have remained non-reacting for at least 11 consecutive years. Similar progress in eradication is also observed in other eastern states as shown in Table II.

TABLE II. Pullorum Disease Testing Summary of 13 States[1] Covering an 11-Year Period

Item	1927-28	1930-31	1933-34	1937-38[2]
Number of tests	735,851	1,565,822	1,597,025	3,076,590
Percentage of positive tests	3.2	3.8	1.2	1.8
Number of negative flocks (100% tested)	372	851	1,120	2,523
Number of breaks in negative flocks (100% tested)	43	85	84	274
Number of accredited flocks	201[3]	377[3]	610	1,197
Number of accredited birds	112,605	240,405	489,836	1,090,076

[1] Connecticut, Delaware, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Virginia, Vermont and West Virginia.
[2] Rhode Island submitted no results for this year.
[3] Massachusetts did not recognize an official accredited grade, which requires a flock to pass two consecutive negative tests not less than six months nor more than one year apart.

Pitfalls in Eradication

Failure to eradicate the disease from a flock is usually the result of following an ineffective program. Too frequently flock owners and hatcherymen place the entire responsibility for eliminating the disease on the macroscopic agglutination test. At best, the test can only detect the carrier of the disease, and the flock owner or testing agency must assume the responsibility of

removing and properly disposing of such reactors as soon as possible. All birds on the premises should be tested. Untested birds should always be regarded as a potential danger even though the flock has been negative for years. Table III shows that flocks which have been non-reacting for several years may become reinfected. Usually the amount of infection in such flocks is not great, but if all birds on the premises had not been tested, it is conceivable that the infected birds might have passed unnoticed. Annual testing of all birds is essential to determine the true status of a flock.

TABLE III. Flock Information on "Breaks"

Explanation for Infection	Number of "Breaks" 1931	1932	1933	1934	1935	1936	1937	1938	Totals	No. of Consecutive Years Non-Reacting Previous to Break 2	3	4	5	6	7	8
Introduction of stock from untested flocks	1					1			2	1					1	
Introduction of stock from tested infected flocks	2	3	1	2					8	4	4					
Eggs hatched at poultry plants with infected flocks	2								2	1	1					
Birds returned from egg-laying contests	1						1		2	1						1
Combination of sources	2	1				1			4	3	1					
Unknown	1	3	6	3	3	3	1	3	23	7	4	3	4	3	2	
Total Breaks	9	7	7	5	3	5	2	3	41	17	10	3	4	3	3	1
Total number of 100% negative flocks the previous year	210	210	180	145	156	163	184	233								
Per cent of breaks	4.29	3.33	3.89	3.45	1.92	3.07	1.09	1.29								

The application of an annual test to an infected flock should not be considered sufficient to stop all losses from pullorum disease. It has been observed in subjecting an infected flock to an intensive retesting program (testing at two- to four-week intervals) that the disease was not eliminated after 15 retests. This flock represents an unusual case in our experience because in the majority of flocks in which infection has been eliminated through retesting, the number of tests to detect all infected birds has been far less, as is pointed out in Table IV. Intensive retesting is advocated only in flocks whose breeding value warrants the expenditure for retesting.

TABLE IV. Retesting Data of Ten Infected Flocks

Flock Number		First Test	Second Test	Third Test	Fourth Test	Fifth Test	Results of Subsequent Season
1	No. of birds	189	152	48			467
	% reactors	1.59	0.00	0.00			0.00
2	No. of birds	369	256	218			232
	% reactors	0.54	0.39	0.00			0.00
3	No. of birds	125	98	91			201
	% reactors	20.00	4.08	0.00			0.00
4	No. of birds	243	262	223	179		199
	% reactors	11.11	1.15	0.00	0.00		0.00
5	No. of birds	464	444	433	397		1.087
	% reactors	2.37	0.45	0.00	0.00		0.00
6	No. of birds	1,765	1,559	1,508	1,108	767	1,796
	% reactors	3.17	0.13	0.00	0.00	0.00	0.00
7	No. of birds	2,079	1,929	1,811	1,648	1,337	2,132
	% reactors	3.17	1.09	0.00	0.00	0.00	0.00
8	No. of birds	704	691	610	422		693
	% reactors	8.24	8.83	0.16	0.00		0.00
9	No. of birds	2,722	2,413	2,284	1,929		3,707
	% reactors	1.80	0.54	0.48	0.00		0.00
10	No. of birds	640	440	399	352	339	747
	% reactors	27.34	4.32	1.00	0.00	0.00	0.00

In the New England states, the most economical method of establishing a pullorum-free flock is through the replacement of the infected flock with stock from officially recognized pullorum-clean sources. The progress in the establishment of pullorum-clean flocks has been made largely through such a procedure in the eastern states. Other states are likewise encouraged to follow this method. However, the hatcheryman and poultryman adopting this scheme of disease elimination should take definite basic precautions against reinfection. Pullorum-clean eggs or chicks should not be permitted to have direct or indirect contact with possible sources of infection. Figure 4 illustrates the very remote possibility of pullorum infection being introduced into a flock and its progeny on a plant as operated by the breeder-hatcher. This mode of operation prevails in the New England states and in some other sections of the country. Figure 5 illustrates the commercial and custom hatchery type of practice in reproducing chicks as it prevails in a great many sections of this country. Commercial and custom hatching is not to be condemned as a means of reproducing chicks. However, the aspect of the practice which cannot be endorsed as sound is the

fact that eggs from infected and non-infected flocks are hatched in the same

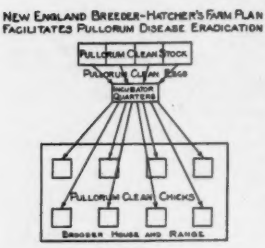

NEW ENGLAND BREEDER-HATCHER'S FARM PLAN FACILITATES PULLORUM DISEASE ERADICATION
PULLORUM CLEAN STOCK
PULLORUM CLEAN EGGS
INCUBATOR QUARTERS
PULLORUM CLEAN CHICKS
BROODER HOUSE AND RANGE

Fig. 4. Schematic illustration of breeder - hatcher plant such as is common in the New England States. Pullorum clean breeding stock, pullorum clean eggs, and progeny all carefully managed by breeder-hatcher.

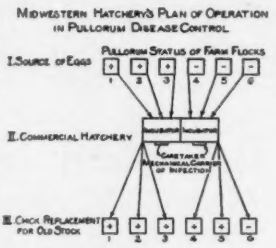

MIDWESTERN HATCHERY'S PLAN OF OPERATION IN PULLORUM DISEASE CONTROL
PULLORUM STATUS OF FARM FLOCKS
I. SOURCE OF EGGS
II. COMMERCIAL HATCHERY
CARETAKER MECHANICAL CARRIER OF INFECTION
III. CHICK REPLACEMENT FOR OLD STOCK

Fig. 5. Schematic illustration of reproducing chicks in some sections of this country. This hatchery selects eggs from various tested supply flocks, some negative and others positive so that the negative flocks are replaced by infected progeny.

hatchery. Little or no progress in eradication can be anticipated through such a practice. Flock owners and hatcherymen should realize that if pullorum-free chicks are desired, one must begin with pullorum-clean breeding stock and not subject the eggs or chicks to possible sources of infection.

The pullorum-clean chicks should not be placed on premises where no precautions are taken against contact with infected mature stock or contaminated shelters or ranges. Figure 6 shows a poultry range which accommodates 10,000 maturing birds, and laying houses for birds after they attain egg production. There is no direct contact between birds on the range and birds in the laying house. This represents the most important break in the dissemination of the infection. Indirect contact between the two groups of birds can be readily established under such facilities if circumstances should demand it. In comparison, Figure 7 illustrates the lack of precautions taken by permitting free contact between young and mature birds, as well as using the same ground year after year. With such

Fig. 6. A modern New England poultry farm with ample range facilities for young birds isolated from the laying quarters which house the adult hens. Cohabitation between young and old stock is avoided.

lack of precautions against reinfection of susceptible stock, one cannot hope to establish a pullorum disease-free flock. Hence the success in maintaining a non-infected

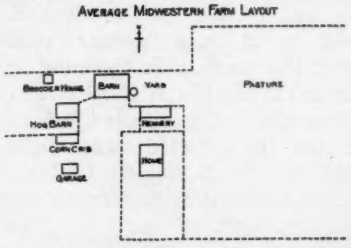


Fig. 7. A schematic illustration of a farm permitting cohabitation of young and old stock—a practice which perpetuates disease.

flock depends upon whether or not the environment for the flock is and remains free from infection. The replacement of infected flocks with pullorum-clean stock should be adopted more generally in our efforts to control and eradicate the disease.

In the testing of flocks for pullorum disease, one should recognize that all reactions obtained do not necessarily represent pullorum infection. In Massachusetts, paratyphoid- and fowl typhoid-infected flocks have been detected in our routine testing for pullorum disease. Furthermore, the detection of only doubtful reacting birds in a previously non-reacting flock should not be sufficient evidence to condemn the flock as pullorum infected. Each year necropsies of doubtful reacting birds detected in previously negative flocks have revealed that when such necropsies do not show infection it appears safe to regard the flock as

negative. The fact is realized that necropsies have their limitations and are not infallible, but to condemn a flock as infected on the basis of doubtful reactions which may be due to causes other than *S. pullorum*, does not seem justifiable. In such instances, it is advisable to supplement the agglutination test with necropsies and bacteriological examination in order to establish an accurate flock diagnosis.

In the control, eradication and prevention of pullorum disease, it should be recognized that the domestic chicken is not the only host susceptible to this infection. Several species of fowl, especially the turkey, have suffered heavy losses from the disease. In many instances, the source of infection is directly or indirectly traceable to infected chickens by way of the incubator or brooder. Pullorum infection in turkeys may follow the same cycle as in chickens (Figure 8). In some sections, the disease has become well established in turkey flocks. Pullorum disease testing of turkeys has been found effective in detecting infected birds, although not to the same degree as with chickens. However, the macroscopic tube agglutination test has been found reliable in detecting turkey flocks free of the disease. In the light of our present knowledge of the disease, it would seem expedient to prevent the incubation and hatching of eggs from untested or infected flocks with eggs from pullorum-passed and pullorum-clean flocks, irrespective of the species of fowl concerned.

Fig. 8. Ovary obtained from a reacting turkey. Numerous misshapen, discolored cystic ova and normal ova were present. Ovarian cultures yielded *S. pullorum*.

National Interest and Education Essential

Interest in pullorum disease control and eradication is rapidly growing in this country. According to a recent report from the United States Department of Agriculture, 42 States during the fiscal year ending June 30, 1939, participated in the pullorum phase of the National Poultry Improvement Plan. However, many of the states recognize only the lowest grade (pullorum tested) of the three grades set up in the plan. Chicks produced from pullorum-tested flocks may on the whole exhibit higher viability than chicks from untested flocks, but losses from pullorum disease are not eliminated as is the case in chicks originating from flocks free from the disease.

Agencies in charge of the control and eradication of pullorum disease in the various states should enlist the coöperative efforts of flock owners, hatcherymen, poultry organizations, county extension services, etc., for educating and encouraging flock owners and hatcherymen to have their flocks and chicks meet the requirements of the two higher grades as recognized by the National Plan. Official lists of pullorum free flocks and hatcheries should be made available to the poultry-buying public. Such lists serve as a very helpful guide in the purchase of stock and in selecting flocks for custom and commercial hatching.

The National Plan for the Control of Pullorum Disease

By HUBERT BUNYEA and
PAUL B. ZUMBRO, Washington, D. C.
Bureau of Animal Industry,
U. S. Department of Agriculture

THE need for organized and official breeding-improvement and pullorum-control programs was recognized in this country as early as 1920. By the year 1925 at least 15 States had initiated poultry-improvement programs on a State-wide basis. The requirements of these programs and the terminology differed, but all had the same purposes—to improve the production and standard qualities of poultry and to reduce losses of baby chicks from pullorum disease.

Many benefits resulted from these State poultry-improvement programs. However, because of conflicting provisions and terminology, the need for a coördinated national plan with uniform provisions and terminology became apparent and was first considered in 1925. Following this many regional and national conferences were held by poultry leaders for the purpose of developing a program that would be applicable to all States. Finally on July 1, 1935, the National Poultry Improvement Plan was placed into operation.

Administration of the Plan

The plan is administered coöperatively by an official State agency in each of the coöperating States and the Bureau of Animal Industry of the United States Department of Agriculture. The official State agency recognized by the Federal Bureau of Animal Industry is usually the agency that was administering the State poultry-improvement program prior to the adoption of the national plan. Among the institutions now serving as official State agencies are State departments of agriculture, State colleges of agriculture, live stock sanitary boards, and poultry improvement boards and associations. In order that poultry improvement boards or associations may be recognized, representatives of one or more of the above State institutions must be on the administrative board. Authority for an official State agency to administer the plan within the State is a memorandum of agreement between the recognized group and the Federal Bureau of Animal Industry. This State agency directs, supervises, and is responsible for flock selection, testing for pullorum disease, and other local administrative work involved in the operation of the plan. The Bureau of Animal Industry is responsible for coördinating the program among the States in which the plan is in operation.

The goal of the National Poultry Improvement Plan with respect to pullorum control is to have practical and efficient pullorum-control programs in every State and to have every industry member participating in the breeding phase of the plan testing his breeding birds under official supervision.

General Features

Some of the general features of the National Poultry Improvement Plan are as follows:

1. The plan is nation-wide in scope. It is entirely voluntary, and any poultry breeder or hatcheryman may participate by signing an agreement with his official State agency and complying with the provisions of the plan.

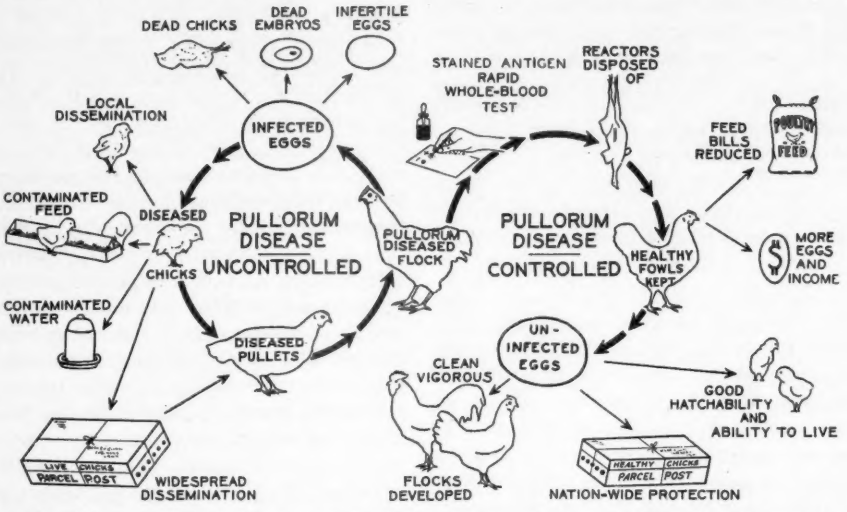
2. All flock-selecting and pullorum-testing work is done by persons authorized by the official State agency.

3. Five progressive breeding stages and three progressive pullorum-control classes are provided.

4. Minimum requirements are established for each breeding stage and each pullorum-control class.

5. Flocks, eggs, chicks, and hatcheries are subject to inspection by official State inspectors who are employes of official State agencies.

6. The official terminology of the plan applies in all parts of the country and is limited to those who are participating in the plan and complying with its provisions.



Comparison of injurious effects of uncontrolled pullorum disease with the benefits resulting from the use of the agglutination test and disposal of reactors. (From Misc. Pub. 349, U. S. Dept. of Agriculture.)

Pullorum-Control Provisions

Pullorum control is one of two major phases of the National Poultry Improvement Plan. The plan provides for the control of this disease since it is known to be passed from the hen to the baby chick through the egg. Furthermore, the losses among baby chicks from pullorum disease may be satisfactorily controlled through the testing of breeding birds by one of the agglutination tests, removing reactors, and using sanitary methods in the operation of hatcheries.

It is a well recognized fact that pullorum disease is for the most part disseminated through the infected ovary of the "carrier" hen, to the egg, and consequently to the chick hatched from that egg. When infected eggs are placed in the commercial incubator, pullorum disease becomes a hatchery problem of the first importance, because infected chicks, their down, droppings, and the egg-shell particles may become very active means of the dissemination of pullorum infection.

The principle of disease control incorporated in the plan is that of the detection of pullorum carriers by means of the agglutination test, and their removal from the breeding flock in order to prevent the introduction of infected hatching eggs into the incubators.

Any one of the following recognized testing methods may be employed under the plan:

1. The standard tube agglutination test, as described in the Proceedings of the United States Live Stock Sanitary Association, November 30 to December 2, 1932, pages 487 to 491.

2. The stained-antigen, rapid, whole-blood test, as described by Schaffer, MacDonald, Hall, and Bunyea, in the *Journal of the American Veterinary Medical Association*, volume 79, No. 2, pages 236-240, 1931, and covered by U. S. Patent 1,816,026.

3. The rapid serum test, as described by Runnels, Coon, Farley, and Thorp, *Journal of*

the American Veterinary Medical Association, volume 70, No. 5, pages 660-662, 1927.

The stained-antigen, rapid, whole-blood method of testing is the most extensively used of the three tests recognized in the plan. The testing methods used by coöperating States during the year ending June 30, 1939, were as follows:

Method	No. States
Stained antigen, rapid, whole-blood	25
Tube agglutination	9
Rapid serum	1
All three	1
Rapid, whole-blood and tube	4
Rapid, whole-blood and serum	2
Total	42

The testing is done by a live stock sanitary authority, an official of the State college of agriculture, or a similarly authorized State employe. However, in the U. S. Pullorum-Tested class the testing may be done by a pullorum-testing agent who has taken a course of training in pullorum testing prescribed by the live stock sanitary authorities or officials of the State college of agriculture and who has passed an examination and been authorized to do the work. Just as in the case of testers employed by the State, the responsibility for the accuracy of the work of the pullorum-testing agents rests with the State authority under whose supervision the pullorum-testing work is carried on.

Three progressive pullorum-control classes are provided in the national plan; namely, U. S. Pullorum-Tested, U. S. Pullorum-Passed, and U. S. Pullorum-Clean. In the U. S. Pullorum-Tested class not more than 10% of reactors may be in the flock at the time of the last test, and these reactors must be removed from the premises of the flock owner before eggs are saved for hatching purposes.

Flocks tested by an official tester and containing no reactors on the last test made within the testing year preceding the date of sale of hatching eggs, baby chicks, and breeding stock, are given the rating of U. S. Pullorum-Passed. To qualify as U. S. Pullorum-Clean, the flock must contain no reactors in either of two consecutive tests not less than six months apart.

It is evident that the success of the pullorum-control program in achieving its goal is dependent upon thoroughness and efficiency in each step of the procedure. The widespread use of the rapid, whole-blood test is at once a challenge and an opportunity for the veterinary practitioner. To the veterinarian who feels that he can devote at least a part of his time to poultry work, the contacts made in connection with testing for pullorum disease should be of inestimable value. He should be in a position to render this important service most competently. At the present time the poultry industry is being victimized by quackery in many forms. The qualified veterinarian is in a position to assume much of the responsibility for the health of the poultry flocks in the United States. He and others may do a great deal to save the poultry industry from the nostrum vender.

In some States all of the pullorum testing is done by qualified veterinarians but in a majority of the States this procedure has not been feasible and laymen have been trained and authorized to do this work. It is desirable to have the birds tested at the same time they are selected for breeding purposes and the general practitioner often finds it difficult, if not impossible, to spend as much time in the field as is required during the testing season. However, as time goes on, and the veterinary profession becomes more active in poultry work, it is conceivable that a much larger proportion of the testing work will be done by veterinarians. It would appear to rest largely with the veterinary profession, as to whether or not this is to come to pass, and how soon.

It is generally recognized that it is desirable to obtain increased services of graduate veterinarians in conducting the pullorum-control work of the National Poultry Improvement Plan. Thus far veterinarians have not participated very much except in the supervision and inspection of pullorum-testing work. Some of the principal reasons reported for failure of veterinarians to par-

ticipate to a greater extent in this program are:

1. Veterinarians have not always shown a sympathetic understanding of general flock management problems. This limits their possibilities of service in this field.

2. It is frequently impossible to obtain the services of veterinarians at times satisfactory to hatcherymen and flock owners to conduct pullorum-testing work.

3. Usually the charges of veterinarians are higher than those made by well trained lay technicians for the same services.

Veterinarians have a wide knowledge of live stock diseases and general sanitation measures which should enable them to render a most valuable service to the flock owners and hatcherymen. It is gratifying to note that veterinarians are familiarizing themselves with the National Poultry Improvement Plan program and taking a greater interest in occupying a logical service position in organized pullorum-control work.

Following the detection of the carrier, comes the necessity for its removal from the breeding flock so that potentially infected eggs may never find their way into an incubator. Pullorum reactors should be sent to slaughter. Their possibilities as future producers of food eggs are never so promising as to justify the risk of keeping them on the poultry breeding farm, as a constant "hotbed" of the dreaded infection. That so much laxity exists in certain quarters in regard to the conscientious disposal of pullorum disease reactors may in great measure be traceable to a lack of scientific knowledge of the danger of harboring diseased fowls on the premises. Here again, the veterinarian, with the traditions of his profession behind him, and his own background of training in sanitary science and animal-disease control, is specially fitted to render signal service to the poultry industry in the control of pullorum disease.

Results to Date

During the first year of the plan 30 States conducted official pullorum-control programs in accordance with its provisions.

TABLE I. States Participating in the Various Pullorum-Control Classes

	1935-36	1936-37	1937-38	1938-39
U. S. Pullorum-Tested	27	32	34	36
U. S. Pullorum-Passed	7	17	14	16
U. S. Pullorum-Clean	8	14	16	19
Pullorum control (total)	30	35	37	42

State participation has steadily increased and 42 States are now coöperating in this phase of the plan. In addition, two States are coöperating in the breeding phase of the plan only. The number of States with U. S. Pullorum-Clean flocks has increased from eight to 19 during the first four years of the plan's operation. During the same period the number of States having U. S. Pullorum-Passed flocks has increased from seven to 16 and the number having U. S. Pullorum-Tested flocks has increased from 27 to 36.

TABLE II. Per Cent of Pullorum-Control Participation in the Various Classes

Classes	1935-36		1936-37		1937-38		1938-39	
	Bird in flocks	% of total	Birds in flocks	% of total	Birds in flocks	% of total	Birds in flocks	% of total
U. S. Pullorum-Tested	1,748,703	85	3,108,919	72	2,859,829	71	5,184,120	77
U. S. Pullorum-Passed	48,967	2	616,929	14	452,217	11	607,143	9
U. S. Pullorum-Clean	261,112	13	585,323	14	724,391	18	980,768	14
	2,058,782	100	4,311,171	100	4,036,437	100	6,772,031	100

It is shown in Table II that the number of birds officially tested in accordance with the provisions of the plan has increased from 2,058,782 during the first year to more than 6,000,000 birds during the fourth year. More than three-fourths of the birds are now in the U. S. Pullorum-Tested class.

During the first year of the plan only 57% of the birds in all phases were officially tested for pullorum disease, but during the last year 78% of all such birds were officially tested.

Incubator Hygiene

By ROBERT GRAHAM and
H. R. HESTER, Urbana, Illinois
University of Illinois

ACKNOWLEDGE of the etiology of pullorum disease as established by Rettger[1] made possible a plan of poultry management, testing of breeding stock, and incubator hygiene in effective control of the disease. These methods in their application have proved valuable and it is recognized that their execution implies a responsibility on breeders, hatcherymen, and live stock sanitary officials. Not the least important phase of the plan is incubator hygiene.

History of Incubator Hygiene

A short time after the etiologic agent of pullorum disease was established, Pernot[2] investigated the disease among incubator hatched chicks, in which the incidence seemed to be much greater than those hatched with the hen. He concluded that if eggs contained pathogenic microörganisms at the time they are laid, the infection within the egg could not be destroyed by disinfection without destroying the vitality of the egg. Incubator disinfection was attempted as an alternative. A small dish of formalin was placed in the incubator until all the chicks were removed. The chicks removed from an incubator disinfected in this manner appeared healthy and the loss from pullorum and other diseases was apparently reduced.

Jones[3] reported the experimental transmission of pullorum disease from artificially infected chicks to healthy chicks in the incubator and brooder. Weaver[4] reported investigations in which 70% of the healthy chicks exposed to infected chicks in the incubator succumbed to pullorum disease. Hinshaw, Upp, and Moore[5] found that pullorum disease was disseminated by artificially infected chick-down placed in a forced-air draught type incubator. Similar results were obtained by infecting the down on hatching chicks and exposing healthy chicks to the down. This down might be carried from the infected to the non-infected chicks. In further studies Hinshaw, Scott and Payne[6] reported the spread of *S. pullorum* in six of eight hatches of chicks. Chicks hatched from eggs of reacting fowls apparently communicated the disease to healthy chicks in the incubator. The mortality in the exposed chicks was greatest in those compartments containing infected chicks and least in the remote compartments of the incubator. Similar results in the communication of pullorum diseases were reported by Bunyea and Hall.[7] They concluded that a large percentage of disease-free chicks succumbed to pullorum disease after exposure in the incubator (without actual control) to the chicks from infected eggs.

Robert Graham

According to Beach and Michael,[8] most outbreaks of pullorum disease originate from infected eggs laid by infected hens. Other sources of the infection include contaminated incubators, shipping boxes, brooder houses, or brooding equipment.

Gwatkin[9] reported fumigating a still-air

[1] Rettger, L. F., 1900. Septicemia among Young Chickens. *N. Y. Med. Jour.* 71:803-805.
[2] Pernot, E. F., 1908. An Investigation on the Mortality of Incubator Chicks. Oreg. Agri. Col. Exp. Sta. Bul. 103.
[3] Jones, F. S., 1910. Fatal Septicemia or Bacillary White Diarrhea in Young Chickens. N. Y. State Vet. Col. Ann. Rpt., pp. 111-129.
[4] Weaver, C. H., 1925. Incubator Infection. Rpt. Dominion of Canada Poultry Husbandman, pp. 56-57.
[5] Hinshaw, W. R., C. W. Upp, and J. M. Moore, 1926. Studies in Transmission of Bacillary White Diarrhea in Incubators. *J.A.V.M.A.* 68:631-641.
[6] Hinshaw, W. R., H. M. Scott, and L. F. Payne, 1928. Further Studies on the Dissemination of Salmonella Pullorum Infection in Incubators. *J.A.V.M.A.* 72:599-610.
[7] Bunyea, H. and W. J. Hall, 1930. Transmission of Pullorum Disease (Bacillary White Diarrhea) in Incubators. *Jour. Agri. Res.* 40:209-223.
[8] Beach, J. R., and S. T. Michael, 1930. Pullorum Disease (Bacillary White Diarrhea of Chickens). Calif. Agri. Exp. Sta. Bul. 486.

type incubator six or eight times without apparent effect upon the hatchability of the eggs. Two cubic centimeters of formalin and 0.66gm of potassium permanganate were employed for each cubic foot of incubator space. Coon[10] sprayed 500cc of formalin into an incubator (1000 cubic feet of air space) without apparent injury to the hatchability of the eggs. The incubator was not disturbed for 1½ hours after the release of the formalin and no attempt was made to close the vents. Dakin and Speer[11] decreased the mortality from 33.2% in the unfumigated incubator to 18% in the incubator fumigated with formaldehyde, and the known spread was reduced from 40% in the unfumigated to 10% in the fumigated incubator.

According to Bushnell, Payne, and Coon[12] the efficiency of formalin as a germicide may be influenced by humidity and rate of diffusion of the gas. Chicks given one short exposure to formaldehyde were apparently unharmed while chicks exposed to larger amounts over a longer period were definitely injured. Since chicks remain in the incubator 36 hours these investigators suggested that continuous fumigation is not feasible during the entire period if injury is to be avoided. However, the hatchability of the eggs was not impaired by the formaldehyde fumigation.

Bushnell and Payne[13] confirmed their earlier investigations and found continuous fumigation by the pan method, as well as the hot plate method of liberation, which severely affects the chicks, to be impractical. The most satisfactory wet bulb reading for disinfection and for hatching of eggs was 88-90° F., while the dry bulb reading was 98-100° F.

Townsley[14] obtained slightly different results with the cabinet type of incubator and a wet bulb reading of 90° F. His results indicate the feasibility of fumigating with formaldehyde (35cc of formalin and 17gm of potassium permanganate per 100 cubic feet of incubator space) at least three times while the chicks are hatching. This treatment did not appear to injure the chicks.

Marcellus, Gwatkin and Glover[15] destroyed pullorum placed on egg shells after 15 minutes of fumigation with 1.5cc of formalin and 1 gm of potassium permanganate, but all embryos between 24 and 96 hours incubation, as well as chicks three hours old, were injured.

In studies made by Graham and Michael[16] it was found that 20cc of formalin per 100 cubic feet of incubator space evaporated from cheesecloth, or 35cc of formalin plus 17.5gm of potassium permanganate per 100 cubic feet of incubator space, consistently killed *S. pullorum* on egg shells and cotton swabs exposed on the inside of an improvised door of the Buckeye Monmouth incubator when the wet bulb reading was 90° F. or more. No differences were noted with open or closed ventilators. However, the location of the contaminated material in the incubator greatly influenced the time required by the formalin to kill *S. pullorum*.

In further studies on incubator hygiene by Graham and Michael,[17] seven different hatches of chicks were subjected to formaldehyde fumigation by the cheesecloth method at two 12-hour intervals, and six hatches by the potassium permanganate method at three 12-hour intervals. No serious injury was noted in chicks fumigated by these methods. Graham and Barger[18] found that fowl-pox virus in a 1% aqueous suspension on cotton squares, or on the feet and the down of day-old chicks, survived 30 to 50 minutes when subjected to formaldehyde released from cheesecloth in amounts of 20cc per 100 cubic feet of incu-

[9] Gwatkin, R., 1926. Some Experiments on the Disinfection of Eggs in the Incubator. Rpt. Ontario Vet. Col., pp. 58-65.

[10] Coon, C. J., 1928. Some Experiments in Disinfecting Incubators with Formaldehyde. *J.A.V.M.A.* 72:627-630.

[11] Dakin, E. L., and F. Speer, 1929. Sanitation in the Hatchery. Ohio State Agri. Col. Ext. Bul. 90.

[12] Bushnell, L. D., L. F. Payne, and C. J. Coon, 1929. Fumigation of Force-Draft Incubators. *J.A.V.M.A.* 75: 611-625.

[13] Bushnell, L. D. and L. F. Payne, 1931. Dissemination of Pullorum Disease in the Incubator. Kan. Agri. Exp. Sta. Tech. Bul. 29.

[14] Townsley, T. S., 1929. Recent Findings Concerning the Proper Temperature and Humidity Conditions for Producing Maximum Hatches of Livable Chicks. Address, Georgia Baby Chick Association Meeting, Macon, Georgia.

[15] Marcellus, F. N., R. Gwatkin and J. S. Glover, 1930. Incubator Disinfection in the Control of Salmonella Pullorum. Conference papers on Disease and its Control, Fourth World's Poultry Congress Rpt., pp. 373-378.

[16] Graham, R., and V. M. Michael, 1932. Studies on Incubator Hygiene I. *Poultry Science* 9:110-116.

[17] Graham, R. and V. M. Michael, 1932. Studies on Incubator Hygiene II. *Poultry Science* 9:197-207.

[18] Graham, R. and E. H. Barger, 1936. Studies on Incubator Hygiene IV. *Poultry Science* 15:48-52.

bator space. However, it was consistently noninfective after 90 minutes.

Graham and Michael[19] found *S. aërytrycke* and *S. newport* nonviable when subjected to formaldehyde fumigation for 60 minutes, while a *Salmonella* sp. isolated from quail survived for 60 minutes, but was nonviable after 90 minutes. Further studies by Graham and Michael on incubator hygiene indicate the advisability of keeping the doors closed on the forced-draft incubator for at least two hours following fumigation, as *S. pullorum* on egg shells survived 100 minutes. Graham and Brandly[20] studied the effect of formaldehyde fumigation upon hemophilic bacteria from cases of coryza, laryngotracheitis virus, and avian bronchitis virus. The hemophilic bacteria and the avian bronchitis virus were found to be nonviable after 60 minutes of fumigation and the same was also true for the aqueous and broth suspensions of laryngotracheitis virus. However, the finely-powdered dried virus was viable in two instances after 90 minutes, emphasizing the necessity for thorough cleaning of the incubator and disinfecting of trays.

Sanitary Measures[21]

Since incubators and incubator rooms in routine operation are subject to the accumulation of dirt, dust, and chick down, frequent cleaning and disinfection must be carefully and systematically carried out if good results are to be obtained. Contamination of the incubator can be minimized by using only those eggs which originate from healthy birds that are maintained in flocks that have been tested repeatedly for several years. Persons who have handled sick chicks should not be allowed to enter the incubator room.

The first essential in incubator hygiene is a clean incubator. Before the hatching season starts the walls and floor of the incubator room should be washed with a reliable disinfectant, and between hatches the egg trays should be carefully cleaned by scrubbing with a steel brush and scalding hot lye water (one pound of lye to 40 gallons of water). After the trays are dry they should be disinfected.

During the hatching season all egg shells, dirt, droppings, and down should be removed from the incubator between the hatches. The down may be removed with a vacuum cleaner.

Any of the following disinfectants may be applied by washing, spraying, or by immersing the hatching trays in a tank:

1. Compound solution of Cresol (Liquor Cresolis Compositus) ..... 4 oz.
   Water ..... 1 gal.
2. Chlorinated lime ..... 4 oz.
   Water ..... 1 gal.
3. Crude carbolic acid..... 7 oz.
   Water ..... 1 gal.

Formaldehyde Fumigation of Incubators

With the formalized cheesecloth method, the relative humidity of the air inside the incubator should be approximately 68% (wet bulb reading 90° F. when the dry bulb reading is 100° F.). Proper humidity reduces the circulation of dust particles and down, favors hatching and facilitates the germicidal action of the formaldehyde. During the time hatching chicks are being fumigated the ventilating fans should be kept running.

For Type A Forced Draft Incubator:

1. Compute the number of cubic feet of air space in the incubator.

2. Cut cheesecloth into pieces approximately one yard square.

3. Place on glass rod in such a position that when the cheesecloth is hung over it the cloth will be as near the fan as possible without interfering with the movement. If the incubator has more than two fans one piece may be used for each fan or a double sized piece hung near one fan.

4. Measure out 20cc (0.67 ounce) of 40% formalin for each 100 cubic feet of incubator space.

5. Dip the cheesecloth into the formalin, using enough cloth to absorb all the formalin

[19] Graham, R. and V. M. Michael, 1936. Studies on Incubator Hygiene. *Poultry Science* 15:494-495.

[20] Graham, R. and C. A. Brandly, 1937. Studies on Incubator Hygiene. *Poultry Science* 16:428-433.

[21] The reader is referred to circular 403, Illinois Agriculture Experiment Station, for a more complete discussion and photographic description of technic to be employed.

[22] Investigations on incubator hygiene conducted with Buckeye Monmouth No. 44 and a Smith Junior.

without allowing the cloth to become wet enough to drip.

6. After all the formalin has been absorbed by the cloth, promptly hang it over the rod and close the door of the incubator.

7. Let the formalized cloth remain in the incubator until at least three hours after the release of the formaldehyde. At this time all dry chicks should be removed.

8. Only two fumigations are necessary during each hatching period when the cheesecloth method is employed. Start the first fumigation when one-tenth to one-fifth of the chicks are out of the eggs, and the second fumigation 12 to 15 hours later.

For Type B Forced-Draft Incubator:

1 and 2. Proceed as directed under 1 and 2 above.

3. Use a piece of cheesecloth large enough to absorb all of the formalin (1½ yards will absorb 100cc). Hang the cloth under one of the fans by means of small wire hooks so that the cloth will be spread as much as possible.

4. Measure out 16⅔cc (0.56 ounce) of formalin for each 100 cubic feet of incubator space.[23]

5. Turn off the fans, allow the cheesecloth to absorb the proper amount of formalin.

6. As soon as the cloth has been treated fasten it under the fan, close the doors and turn on the fans.

7 and 8. Proceed as in 7 and 8 above.

Potassium Permanganate Method

The same conditions of humidity, temperature, and cleanliness as required for fumigation with cheesecloth are employed for releasing formaldehyde with potassium permanganate.

For Forced-Draft Incubator Type A or B:[24]

1. Determine the number of cubic feet of incubator air space.

2. For each 100 cubic feet of incubator space use 35cc of formalin (1.17 ounces) and 17.5gm (0.6 ounce) of potassium permanganate.

3. Place the calculated amount of potassium permanganate in an enamel pan large enough to hold 10 times the quantity of material used. Do not use glass or earthenware containers, as the heat generated by the chemicals in combination may cause them to break.

4. Set the pan in the center of the incubator below the fan; for large incubators more than one pan may be necessary. In some force-draft incubators the materials may be placed in a broad, shallow pan which can be set in the cabinet under the intake pipe. Leave the damper of the intake pipe wide open so that the gas can pass freely into the incubator.

5. Pour the computed amount of formalin over the permanganate crystals and close the door as quickly as possible. The formaldehyde gas is liberated immediately.

6. Three fumigations 12 hours apart should be made during the hatching period. The first fumigation should be made shortly after the first chicks come out of the egg. In an experiment conducted at the Illinois Experiment Station a few chicks subjected to the three fumigations manifested symptoms of inanition. However, they were of a transitory nature. The number of chicks that have died from the effects of fumigation has not been important, as the majority of the fatalities occurred among subnormal chicks.

Fumigation of Contaminated Eggshells and Cotton

S. pullorum was placed on cotton squares (1"x1"x1/16") and eggshells, which were later placed in different locations in the incubator. These contaminated materials were removed from the incubator at 5- to 30-minute intervals and cultured on suitable media. The results suggest that the location of *S. pullorum* in the incubator may be a factor which greatly influences the time required for the formaldehyde gas to destroy the infection.

When *S. pullorum* was exposed on cotton squares or eggshells attached to the inside of a false door of the incubator so that the fumigant had access to all sides of the contaminated material, it was killed much more quickly than when the material was placed in an open petri dish set in the hatching tray or on the floor of the incubator.

Likewise, the nature of the contaminated material seemed to influence the resistance of *S. pullorum* to the fumigant. The organism was killed more quickly on eggshells than on cotton squares. This may be explained by the fact that formaldehyde gas does not readily penetrate the cotton

[23] A total of 180cc should be used for the Smith Senior incubator and 100cc (3.3 ounces) for the Smith Junior incubator.

[24] Smith or Buckeye Mammoth.

squares. Droplet or air infection was most rapidly destroyed, while contaminated cotton squares afforded the greatest resistance to fumigation. Ten fumigations with formaldehyde gas by the potassium permanganate method were made in one incubator. In one of the ten, *S. pullorum* on cotton squares in the egg trays was alive at the end of 180 minutes, while in the remaining nine fumigations, as well as in the 10 fumigations with formalized cheesecloth, *S. pullorum* was consistently killed within 180 minutes. In 25 fumigations with potassium permanganate in another incubator and in 25 with formalized cheesecloth, *S. pullorum* on contaminated cotton squares was consistently killed within 180 minutes.

Fumigations of artificially contaminated cotton squares placed on egg trays and hatching trays of two forced-draft incubators yielded results comparable to those obtained in the fumigation of artificially contaminated chicks. These observations suggest the necessity of keeping incubators free of organic materials (eggshells, down, droppings, and dead chicks) to obtain efficient fumigation.

Fumigation of Artificially Contaminated Chicks

Chicks hatched from infected eggs are sometimes covered with a suspension of *S. pullorum*, and as the down dries and fluffs out, the organism may be carried by the incubator air currents from the infected chicks to disease-free chicks. The chief purpose of fumigating incubators is to kill the disease-producing bacteria at hatching time before healthy chicks become infected.

In an effort to determine the efficiency of formaldehyde fumigation in destroying natural *S. pullorum* infection on chick feet and down, healthy day-old chicks, following an immersion in a suspension of *S. pullorum*, were placed in the hatching trays of two forced-draft incubators and fumigated with formaldehyde. The formaldehyde gas was released by means of the potassium permanganate and from formalized cheesecloth. The artificially contaminated chicks were examined for living *S. pullorum* at intervals of 30 minutes for a period of three hours. After the release of the fumigant, *S. pullorum* survived on the feet and down of the chicks exposed to the gas for periods ranging from less than 30 minutes to more than 180 minutes. However, in the majority of fumigations, the organism was found nonviable in less than 60 minutes. The average lethal time of the different fumigations for *S. pullorum* ranged from 57 to 93 minutes.

The results of fumigating artificially contaminated chicks suggest that formaldehyde gas released in forced-draft incubators in amounts recommended by manufacturers may not always destroy *S. pullorum* on the feet and down of infected chicks within three hours, since in one of the forced-draft incubators two of 22 fumigations by the potassium permanganate method failed to destroy the organism within 180 minutes. Twenty fumigations of the same incubator with formalized cheesecloth consistently destroyed *S. pullorum* on the feet and down of chicks artificially infected within 180 minutes. In the other forced-draft incubator *S. pullorum* on the feet and down of chicks did not survive as long as 180 minutes in any of the 10 fumigations conducted. Both methods for releasing the formaldehyde gas were employed in this experiment and similar results were obtained in all instances.

Influences of Fumigations on Chick Mortality

No noticeable injury to healthy chicks resulted from fumigations at the time of hatching. Weak chicks were sometimes injured, but this is a negligible factor and is more than overbalanced by the protection which fumigants accomplish in preventing the spread of disease in incubators. It is readily apparent, however, that chicks developing from infected eggs cannot be cured by fumigation.

The percentage of survival among the chicks of 15 hatches fumigated with formalized cheesecloth was, after the first two weeks, five points higher than the per-

centage of survival among the chicks of three unfumigated hatches. The 15 fumigated hatches included 2,305 chicks, of which 13% died during the first two weeks. The three unfumigated hatches included 397 chicks, of which 18% died during the same period.

The part which pullorum disease played in the chick mortality of these hatches was suggested by the direct isolation of *S. pullorum* from 7.56% of the chicks that died in the control hatches and from 4.47% of the chicks that died in the fumigated hatches. Eggs from the same flock were used in both the control and fumigated hatches. *S. pullorum* was isolated in approximately 50% of the dead embryos in both hatches.

Disinfecting Values of the Two Methods of Fumigation

The results of these experiments indicate that either the potassium permanganate or the formalized cheesecloth method of fumigation of incubators, if properly performed, is valuable in reducing the dissemination of pullorum disease. The method to be employed is optional; each hatcheryman will probably obtain the best results with the method he can employ most accurately. It is not safe to assume that the results of fumigation may be precisely the same in different incubators unless the rate of air change, humidity, temperature, and physical cleanliness are the same. Incubator fumigation is regarded as a relative procedure, having a maximum efficiency in clean incubators in well ventilated incubator rooms.

Double Fumigation Before Hatches

Twenty-four hours before each hatch it is advisable to give the incubator a double strength fumigation to destroy latent or mass infection. The double strength fumigation may also be used to disinfect cleaned incubators at the start of the hatching season. Eggs are not injured by repeated fumigation with double amounts of formalin, but chicks should not be exposed to this concentration. Healthy chicks tolerate amounts of formaldehyde gas that possess distinct germicidal properties for *S. pullorum*, but chicks which have a low vitality or chicks that harbor the infection may be injured by exposure to routine fumigations. Chicks more than 48 hours old should not be subjected to formaldehyde fumigation.

Summary

1. Formaldehyde fumigation of clean forced-draft incubators by either the cheesecloth method or the potassium permanganate method is of definite value in preventing the dissemination of *S. pullorum* through the incubator.

2. Clean incubators and incubator rooms also aid in controlling pullorum disease.

3. Hatching trays should be scrubbed and disinfected after each hatch.

4. The fumigation of incubators may be carried out during the hatching period.

Animal Pathology Laboratory, University of Illinois

Nematodes of Domestic Fowls Transmissible to Wild Game Birds

By EVERETT E. WEHR, Washington, D. C.
Associate Zoölogist, Bureau of Animal Industry, U. S. Department of Agriculture

BREEDERS of wild game birds have been forced to abandon the practice of brooding their chicks under bantam hens because they soon learned that the young chicks frequently became infected with the same diseases and parasites that were present in the foster mothers. The young birds acquired these diseases and parasites by ingesting food contaminated with the droppings of the infected foster mother hen, or by eating insects, snails, and other intermediate hosts infested with the immature stages of nematodes, cestodes, etc.

The damage due to worm parasites is not so spectacular as is that due to such protozoan diseases as blackhead and trichomoniasis; however, the total loss occasioned by worm parasites is frequently just as great as that produced by the protozoan diseases mentioned. Worm parasites, especially nematodes, have been found to be abundant, both as to numbers and species, in wild game birds, and many of these have been found to be identical with those parasitizing domestic fowl.

In the following pages the author has attempted to discuss in a general way the principal nematodes common to domestic and wild fowl and has pointed out the danger of rearing the two types of birds together, or even in close proximity to each other.

Gapeworms

The gapeworm, *Syngamus trachea*, apparently is the cause of considerable losses among pheasants, but, so far as we are aware, it is of little importance as a parasite of other wild game birds in the United States. Worms identified as this species have been collected from the wild turkey, red grouse, ruffed grouse, duck, and quail. It is possible, however, that the worms recovered from these different hosts may represent more than a single species. Among the domestic fowl, this nematode is particularly injurious to chicks, turkey poults and goslings. The guinea fowl is susceptible to gapeworms at any age but does not develop clinical symptoms of infection. The worms may be the source of considerable annoyance to adult birds, but seldom cause death, except in cases of heavy infestations.

The adult worms live in the trachea where they attach themselves to its lining and suck blood. The male and female worms are permanently joined together so that the pair has the appearance of a single worm shaped like the letter Y. The female worm is much larger and is usually redder in color than the male, the color being due to the larger amount of blood extracted by the female. Since the male worm apparently remains attached to the tracheal wall throughout most of its existence, it is believed that it serves as an anchor for the female and permits her to detach and reattach for the purpose of feeding. Nodule formations in the trachea are frequently observed at the point of attachment of the male worms.

Gapeworms affect young birds primarily. An extensive irritation of the mucous lining of the trachea is produced as a result of the presence of these bloodsucking worms. Coughing is apparently the result of this irritation. Gaping quickly follows coughing, which is usually accompanied by a quick jerk of the head forward and upward and an extension of the neck, indicating that the bird is not getting a sufficient amount of air. Sudden deaths are due primarily to suffocation caused by mechanical obstruction of the trachea by the rapidly growing worms and the rapid accumulation of mucus.

The eggs of the female worm are set free

in the lumen of the trachea, are coughed up, swallowed, and passed to the exterior in the droppings. Active embryos develop inside these eggs in from nine to 14 days, depending on such factors as temperature and moisture. Such eggs hatch when taken into the alimentary tract of a susceptible bird host. The larvæ then penetrate the wall of the intestinal tract and pass to the lungs. Here the larvæ feed for several days, developing to the immature adult stage. It is in the lungs that the male and female unite in permanent copula. Thus united, the male and female worms migrate up the bronchi into the windpipe. From the time the larvated eggs enter the body of the bird until the copulated pair is first observed in the trachea, a period of seven days has elapsed, and seven or eight days more are required for the worms to reach egg-laying maturity.

Susceptible birds may also acquire gapeworms by ingesting earthworms that have fed on soil contaminated with the droppings of infested birds.

Experiments conducted by the Bureau of Animal Industry indicate that barium antimonyl tartrate used as a dust is an effective drug for the removal of gapeworms from chicks and turkey poults. The birds are exposed to the chemical for 15 to 20 minutes in a closed container or box, the size of the dose being determined by the capacity of the box. One ounce of the finely powdered dust is sufficient for a box having a capacity of eight cubic feet. The box or container should be deep enough to permit the birds to stand erect and just enough birds placed in the box to fill it about two-thirds full. The dust is introduced by means of a dust gun through a hole in the side of the box.

Crop Worms

The crop worm, *Capillaria annulata*, occurs in tortuous burrows in the mucosa of the crop and esophagus of the bobwhite quail, turkey, pheasant, ruffed grouse, Hungarian partridge, and chicken. With the exception of the chicken and the turkey, and possibly the quail, this nematode has been found only occasionally in the above hosts.

A pitfall in rearing wild game birds is permitting them to associate with domestic fowl and thus acquire serious and even fatal parasitisms. Among the nematodes that may be communicated in this way from barnyard fowl to wild game birds are: gapeworms and crop worms, acquired by quail from chickens and turkeys; stomach worms, by grouse, quail, pheasants and pigeons from guinea fowl, turkeys, and chickens; and intestinal worms, including ascarids, trichostrongyles, Heterakis and Capillaria, acquired by grouse, prairie chickens, quail and doves from various domestic birds.

The chief injury produced by the crop worm is a thickening of the wall and an enlargement of the glands of the crop and esophagus. In heavy infections, the crop wall is greatly thickened and the mucosa is loose and torn. Allen and Gross[1] reported severe anemia in a ruffed grouse which had died as a result of a heavy infestation with *Capillaria annulata*.

Wehr[2] demonstrated that under both natural and artificial conditions two species of earthworms, *Helodrilus fœtidus* and *H. caliginosus*, served as intermediate hosts for *C. annulata*.

Another species of crop worm, *Capillaria contorta*, occurs naturally in the bobwhite quail, domestic duck, turkey, and pheasant, but this worm has never been encountered in chickens. The damage produced by this parasite is similar to that observed for *C. annulata*. Unlike *C. annulata*, this capillarid does not require an intermediate host.

There is no satisfactory treatment for the

[1] Allen, A. A. and A. O. Gross, 1926. Ruffed Grouse Investigations. *Bull. Am. Game Protect. Ass.*, 15:4, pp. 81-86.

[2] Wehr, E. E., 1936. Earthworms as Transmitters of *Capillaria annulata*, the Crop Worm of Chickens. *North Amer. Vet.*, 17:8, pp. 18-20.

removal of crop worms. Prevention involves strict sanitation. The practice of rearing birds on well-drained soil in sections where earthworms are abundant will materially reduce infections with *C. annulata* since earthworms are never found where the soil is dry. Manure should not be allowed to collect in the poultry yards, as accumulation of droppings will tend to attract earthworms. Earthworms are greatly relished by many species of birds.

Stomach Worms

The spiral stomach worm, *Dispharynx spiralis*, has been found to be a parasite of the grouse, Hungarian partridge, bobwhite quail, pheasant, pigeon, guinea fowl, turkey, and chicken. Yeatter[3] found the incidence of the parasite among Hungarian partridges in the Great Lakes region to be 31.6%. Boughton[4] did not mention this species of worm as occurring in grouse in Minnesota. *D. spiralis* is most prevalent among wild game birds in the New England states and New York. A large percentage of the grouse found dead or in a weakened or dying condition that were examined by the New England Grouse Investigation Commission were infected with this parasite. Bump[5] stated that this worm was the most important parasite recovered from wild game birds in New York, and Allen[6] expressed the belief that *D. spiralis* was the chief cause of "grouse disease" in northeastern United States. Heavy infections of this parasite resulted in the death of many carrier pigeons of the Signal Corps of the United States Army, Fort Sam Houston, Texas, a few years ago, and wild pigeons caught in and around Balboa Park, San Diego, California, and examined in the Bureau of Animal Industry were found to be heavily infested with this parasite.

This nematode occurs in the glandular stomach of its host and may be the cause of marked pathological changes. The worms burrow deeply into the mucosa of the proventriculus, causing the formation of tumors at the site of infection. In heavy infections, the wall of the proventriculus becomes greatly thickened and ulcerated. Allen and Gross[1] described the pathological changes in the stomach of ruffed grouse due to *D. spiralis* as follows: "Tunica propria frequently found swollen to twice its normal size. In extremely severe cases of infection, the parasites get down into the deep gastric glands and the general appearance of the proventriculus when opened is that the whole interior of the organ has been macerated and reduced to shreds. Tissue layers are indistinguishable and the parasites are found on the surface and hidden between and underneath the proliferated tissues. Such infestations are accompanied by an abnormal secretion of mucus."

Cram[7] demonstrated that the pill bug, an isopod, served experimentally as the intermediate host of the spiral stomach worm. Cultures of eggs of this parasite collected from ruffed grouse were used as the infective material in these experiments. Quail which were fed some of the infected pill bugs later became infected with the adult nematodes. This same crustacean also served experimentally in the transmission of this parasite from pigeon to pigeon. Whether this isopod serves as the principal intermediate host for the spread of this parasite under natural conditions is not known.

Since there is no satisfactory drug known for the removal of this nematode, measures which will serve to control or eradicate the intermediate host from the premises are indicated. Sanitation of the premises and the proper disposal of droppings so that the birds cannot gain access to them at any time are important.

Tetrameres americana, a nematode strikingly different in appearance from most roundworms, occurs in the glandular, or true stomach, of the chicken and bobwhite quail. The male of this nematode is very

[3] Yeatter, R. E., 1934. The Hungarian Partridge in the Great Lakes Region. Bull. (5), Univ. Michigan School Forestry and Conserv.

[4] Boughton, R. V., 1937. Endoparasitic Infestations in Grouse, their Pathogenicity and Correlation with Meteorotopographical Conditions. Univ. of Minnesota, Agric. Exper. Station, Technical Bulletin No. 121.

[5] Bump, G., 1935. Ruffed Grouse in New York State during the Period of Maximum Abundance. Trans. Amer. Game Confr. 21, pp. 364-369.

[6] Allen, A. A., 1925. The Grouse Disease in 1924. *Bull. Amer. Game*, 14:3, pp. 11, 12, 30.

[7] Cram, E. B., 1931. Developmental Stages of Some Nematodes of the Spiruroidea Parasitic in Poultry and Game Birds. Techn. Bull. (227), U. S. Dept. Agric.

small and resembles other nematodes in appearance. It lives free in the lumen of the proventriculus. The female is globular in shape, bright red in color, and is found in the glands of the stomach wall, with only the tail protruding into the lumen of the proventriculus. Cram[8] found this parasite to be common in quail in Virginia which had been raised in captivity and in close proximity to poultry. Stoddard[9] reported *Tetrameres americana* as occasionally being found in quail under natural conditions in the southeastern United States. Barber[10] stated that the glandular stomach worm was the cause of a serious catarrhal condition in chickens in Guam. In cases of heavy infestations, the walls of the proventriculus became so swollen that the lumen was almost obliterated.

An intermediate host is required for the complete development of this roundworm. Cram[7] fed embryonated eggs of this worm to grasshoppers, *Melanoplus femurrubrum* and *M. differentialis*, and recovered infective larvæ from the body cavities of these insects approximately 42 days after infection. These larvæ were fed to a number of bird hosts and infestations were obtained in chickens, in the domestic duck (one female specimen obtained from a young duck), bobwhite quail, ruffed grouse, and pigeon. It is assumed that the bobwhite quail to which these eggs were fed were young birds because the same author in another publication stated that she was unable to infect with *Tetrameres americana* two quail that were approximately five months old.

No satisfactory treatment other than strict sanitation is known for the control of this parasite. Due to its protected position in the glands of the proventriculus, the female would be difficult to destroy with a drug that would be harmless to the host.

Another species of tetramere, *Tetrameres fissispina*, has been reported from wild and domestic ducks and chickens in Europe. Sugimoto and Nishiyama[11] stated that this stomach worm was fairly common in chickens in Formosa and was of considerable pathological importance. Heavily infested birds were emaciated and anemic.

The life history of *T. fissispina* has been found to involve the amphipod, *Gammarus pulex*, the cladoceran, *Daphnia pulex*, grasshoppers, cockroaches, and earthworms.

The small, slender roundworm, *Amidostomum anseris*, has been reported from wild and domestic ducks and geese. This parasite occurs underneath the horny lining of the gizzard and sometimes in large numbers. For many years, this parasite has been reported as the cause of losses among domestic geese in Europe. Cram[12] reported for the first time an outbreak of amidostomiasis in the United States. This outbreak occurred among a flock of geese in New York, the owner of this flock reporting a large number of deaths as a result of this infestation.

Young birds are especially affected, dullness, loss of appetite and emaciation being the chief symptoms of infestation. The clinical symptoms are largely the result of the improper functioning of the diseased gizzard. On post-mortem, the inner surfaces of heavily parasitized gizzards appear necrotic. The horny lining is loosened or sloughed in places and appears dark brown or black in color at the site where the worms are located.

The life history of this nematode does not involve an intermediate host. The eggs pass out in the droppings and the young worms which hatch from them become infective within a few days. When the infective larvæ are picked up by a susceptible host, they develop to the adult stage in the gizzard.

Carbon tetrachloride has been found to be an effective remedy for the removal of

[8] Cram, E. B., 1931. Internal Parasites and Parasitic Diseases of the Bobwhite. Nematodes (Roundworms) in Quail. (*In* Stoddard, Herbert L., "The Bobwhite Quail; its Habits, Preservation and Increase," pp. 240-296.)

[9] Stoddard, H. L., 1931. "The Bobwhite Quail; its Habits, Preservation and Increase."

[10] Barber, L. B., 1916. Live Stock Disease Investigations. Ann. Report Guam Agric. Exper. Station (1915), pp. 25-41.

[11] Sugimoto, M., and S. Nishiyama, 1937. On the Nematode, *Tropisurus fissispina* (Diesing, 1835) and its Transmission to Chickens in Formosa. *J. Soc. Trop. Agric.* (Nettai Nogaku Kwaishi), Taihoku Imp. Uni., 9:2, pp. 226-235.

[12] Cram, E. B., 1926. A Parasitic Nematode as the Cause of Losses among Domestic Geese. *North Amer. Vet.*, 7:1, pp. 27-29.

A. anseris. The drug may be given in capsules or mixed with liquid cereal or flour paste, the amount per bird being 1.5 to 2cc.

Seurocyrnea colini is of common occurrence in the gizzard of the bobwhite quail of the southeastern states. It has also been collected from turkeys (presumably wild turkeys) in Georgia, prairie chickens in Wisconsin, and sharp-tailed grouse in Wisconsin and Montana. Experimental infestations have been produced in the chicken and in the quail. There has been little or no pathological change observed in connection with this parasite.

S. colini requires an intermediate host for its complete development. Cram[7] produced experimental infestations in the chicken and the quail by using the cockroach, *Blatella germanica*, as the intermediary.

There is no satisfactory treatment known for this stomach worm.

Intestinal Worms

The large intestinal roundworm of chickens, *Ascaridia galli*, has been reported to be the most common nematode parasite found in the intestines of the sharp-tailed grouse, prairie chicken, and ruffed grouse in the United States and Canada. There are also records of this nematode from the bobwhite quail, domestic goose, domestic duck, chukar partridge, turkey, and other birds. The fact that this ascarid has been reported on numerous occasions from a wide variety of birds, both domestic and wild, prompted the writer to undertake a comparative study of a large number of specimens of ascarids collected from wild game birds, particularly the grouse, in order to determine if they were the same or a different species than that commonly found in domestic fowl. This study, although still incomplete, has progressed sufficiently to show that a number of differences exist between the grouse form and the chicken form. However, a more detailed study of the grouse form is necessary to determine its proper systematic disposition. Although Boughton[4] definitely identified the grouse ascarid as *A. galli*, he stated that the grouse form differs from the chicken form in the arrangement of the papillæ on the tail of the male. However, he regarded these differences as variations only. The same differences between the two forms, as noted by Boughton, have been consistently observed by the writer.

So far as is known, very little cross-infection work has been done with the ascarids of either domestic or wild game birds. Ackert and Eisenbrandt[13] found that turkeys were much more resistant to infestation with *A. galli* than were chickens under experimental conditions. The present writer was unsuccessful in infesting partially-grown and full-grown domestic turkeys with *A. galli* from the chicken. Cram[8] reported having found quail naturally infested with immature specimens of this nematode. However, she was unsuccessful in experimentally infesting adult quail by administering larvated eggs of *A. galli*.

Ascarids normally reside in the small intestines of birds, but on several occasions investigators, notably Gross[14, 15] and Allen and Gross[1] have reported the grouse ascarid as invading the body, burrowing into the flesh of the breast, or into the region behind the lungs or kidneys. According to these authors, this worm, when outside its normal habitat, may cause serious injury. Gross[16] stated that young birds were seriously affected by infestations with this ascarid.

The life history of *A. galli* is direct. The eggs are set free in the intestine of the host and pass to the exterior in the droppings. Under favorable conditions of temperature and moisture, the eggs contain active embryos within 10 days to two weeks. Clean birds become infested with this parasite by ingesting larvated eggs with the food and water.

In the domestic fowl, the removal of this worm is most successfully accomplished by

[13] Ackert, J. E. and L. Eisenbrandt, 1933. On the Comparative Resistance of Bronze Turkeys and White Leghorn Chickens to the Nematode *Ascaridia lineata* (Schneider). Program, 9th Ann. Meet. Am. Soc. Parasitol., Boston, Dec. 28-30. *J. Parasitol.* 20:2, p. 129.

[14] Gross, A. O., 1928. Progress Report of the New England Ruffed Grouse Investigations Committee. Massachusetts Fish and Game Association, Boston.

[15] Gross, A. O., 1930. Progress Report of the Wisconsin Prairie Chicken Investigations. Wisconsin Conservation Commission.

[16] Gross, A. O., 1933. New England Ruffed Grouse Investigations. Tr. 19. Amer. Game Confr. New York, pp. 466-467.

treating each bird individually with carbon tetrachloride or tetrachlorethylene, the dose being 1cc for adult birds. Treatment of young birds is considered to be risky and should not be practiced as a regular procedure.

The strongyle, *Trichostrongylus tenuis*, has been reported from the domestic and wild duck, domestic and wild goose, pheasant, chicken, turkey, guinea fowl, grouse, and quail. Several years ago, this species was reported as one of the principal causes of deaths among grouse in Europe. An outbreak of trichostrongylosis due to this parasite occurred among young domestic geese in the district of Columbia a few years ago. The worms were present in large numbers in the affected birds and no doubt contributed to the high mortality that occurred among the birds of this flock. Since these birds were also suffering from renal coccidiosis, the trichostrongyles could not be held solely responsible for the total number of deaths. According to Cram[8] this threadworm is of wide distribution among quail of the southeastern United States.

Trichostrongylus tenuis occurs in the ceca and when present in large numbers, such as have been found in the red grouse of Europe, it produces definite clinical symptoms and lesions. The changes in the ceca consist of a thickening and reddening of the walls, and small hemorrhages are sometimes present. Loss of weight, anemia, and chronic toxemia have been reported as symptoms resulting from heavy infestations.

The life history of this parasite is direct. The eggs hatch very soon after they have been expelled in the cecal contents. Within approximately two weeks, the freshly hatched larvæ have molted twice and have become infective. In this stage they are very resistant to cold. When picked up by a susceptible host the infective larva molts twice more within the ceca of the bird before reaching the adult stage.

T. tenuis from pheasants have been successfully transmitted to the domestic turkey, guinea fowl and chicken.

There is no known treatment for this parasite other than sanitation.

The cecal worm, *Heterakis gallinæ*, has been reported from the ceca of wild turkeys, pheasants, ruffed grouse, quail, sage grouse, and such domestic fowls as the turkey, chicken and guinea fowl. Our knowledge of the occurrence of this species in wild game birds is very incomplete, since it is believed that this species has been confused with other species of the same genus occurring in the same hosts. Boughton[4] stated that *H. gallinæ* was very rare among ruffed grouse in Minnesota, only 0.62% of the grouse examined being infested. Canavan[17] reported *H. gallinæ* from the golden pheasant and the bobwhite quail in the Philadelphia Zoölogical Garden. The description accompanying this report is obviously that of another species of cecal worm, probably *Heterakis bonasæ*, described by Cram in 927 from the ruffed grouse in New York. Cram reported *H. gallinæ* from the bobwhite quail raised in captivity and in close proximity to domestic fowl.

No recognizable gross pathological changes in the ceca have been observed as a result of an infestation with these worms. Birds that are heavily infested with cecal worms are sometimes unthrifty. In the chicken and turkey, and possibly in other birds, *H. gallinæ* plays an important part in the spread of blackhead disease, although it is not an essential factor in the dissemination of this disease. The blackhead organisms may pass out of the bird's body enclosed in the eggs of the cecal worm and, protected by the thick shell of the egg, they are able to resist adverse climatic conditions more effectively than they would otherwise.

Another species of cecal worm, *H. bonasæ*, has been reported as stated above, from the ceca of ruffed grouse and from the bobwhite quail in Massachusetts and southeastern United States; it has been found to be especially abundant in quail from Georgia and Florida.

A third species of cecal worms, *Heterakis*

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[17] Canavan, W. P. N., 1929. Nematode Parasites of Vertebrates in the Philadelphia Zoölogical Garden and Vicinity. I. *Parasitology*. 21:1, 2, pp. 63-102.

[18] Schwartz, B., 1924. Occurrence of Nodular Typhlitis in Pheasants due to *Heterakis isolonche* in North America. *J.A.V.M.A.*, 15:5, pp. 1-7.

isolonche, was reported from pheasants in Pennsylvania and Connecticut by Schwartz[18]; these nematodes occur in cecal nodules. Death may result from an infection with this parasite. Letulle and Marotel[19] reported two deaths, and Lucet and Henry[20] reported six deaths, among pheasants from nodular typhlitis in France.

The life history of *H. gallinæ* as worked out in the domestic fowl is direct, and it is likely that the life histories of *H. bonasæ* and *H. isolonche* are similar. Earthworms have been reported as playing a rôle in the spread of the eggs of *H. gallinæ* from place to place.

There is no drug that may be added to the mash that will satisfactorily remove a large proportion of these parasites from infested birds. Individual treatment is the only logical procedure to follow. This consists in the use of rectal injections of oil of chenopodium in a bland oil, such as cottonseed oil. In case of the domestic fowl, the dose for a bird weighing one and a half pounds is 0.1cc of oil of chenopodium in 5cc of cottonseed oil. This treatment has removed as high as 90% of the worms from a single bird. This mixture may be made up at the rate of one teaspoon of oil of chenopodium in six fluid ounces of cottonseed oil and given at the rate of one-third of an ounce to birds weighing three pounds or more. The two ingredients should be thoroughly mixed and given with a hard-rubber enema syringe.

There are two other nematodes which are occasionally found in both domestic fowl and wild game birds. The pigeon strongyle, *Ornithostrongylus quadriradiatus*, occurs in the small intestines of domestic and wild pigeons and the mourning dove and may be of serious pathological significance. These bloodsucking worms may cause extensive destruction of the intestinal mucosa, sometimes accompanied by an abundance of bright green stained mucus. In severe cases of parasitism extensive hemorrhages are also in evidence. The control of this parasite can best be accomplished by destroying the young preparasitic stages; this may be accomplished by locating the pigeon lofts on well-drained soil having sunny exposures.

The pigeon capillarid, *Capillaria columbæ*, is normally a parasite of the small intestine of the domestic and wild pigeon. However, this nematode has been reported from the intestines of the mourning dove, chicken, and turkey. Birds heavily infested with *C. columbæ* show definite symptoms of emaciation, listlessness, and diarrhea, and death may occur in case of heavy infestations. The intestines show an extensive destruction of the mucosa, frequently with complete sloughing of the mucous membrane. The control of this parasite should be directed toward prevention by thorough sanitation and selection of well-drained areas for permanent lofts and houses. Chickens, turkeys, and pigeons should be raised separately, since all three types of birds are susceptible to *C. columbæ*.

Conclusions

The most important nematodes common to both domestic and wild game birds have been discussed. A survey of the available literature dealing with the occurrence of these parasites in both types of birds strongly suggests that further and more careful work is needed to clarify many statements concerning these parasites. We need, first of all, to acquaint ourselves better with the species affecting wild game birds in order to establish their possible identity with those occurring in domestic fowls. Several nematodes found normally in wild game birds have been reported as species common to domestic birds; yet the descriptions accompanying these reports are obviously those of other and related species. There is, nevertheless, sufficient accurate knowledge concerning the occurrence of nematodes of domestic birds as parasites of wild game birds and the possibility of cross-infection, to warrant breeders of wild game birds to avail themselves of this information in order to be able better to avoid some of the pitfalls that have caused many beginners to abandon raising game birds under conditions of domestication.

[19] Letulle, M., and G. Marotel, 1909. Etude des Typhlites Parasitaires. Nodules des Cæcums parasitaires chez le Faisan. *Arch. de Parasitol.*, 12:3, pp. 361-368.

[20] Lucet, A., and A. Henry, 1911. La Typhlite Verrugueuse des Faisans et Son Parasite (*Heterakis isolonche* v. Linstow) *Bull. Soc. Centr. de Méd Vét.*, pp. 320-333.

The Gestation Period in the Bitch

By LEON F. WHITNEY,
Auburn, Alabama
Alabama Polytechnic Institute, Class of 1940

HOW long is the gestation period in the bitch? Various estimates have been given, but none, so far as the author is able to determine, has been the result of clear-cut records. That normal estrous periods have great variation when judged superficially, no one can deny. From among the many hundreds of matings which have resulted in over 5,000 puppies born in the author's kennels, there are 30 which resulted in puppies when the dogs were allowed to copulate only once. An analyis of these matings, together with others, may help to determine more accurately the answer we are seeking to this problem.

In studying the length of the gestation period, little information can be obtained from pregnancies which have resulted from one of many matings, since it cannot usually be ascertained which was the effective mating. It is, therefore, perhaps fortunate that even as few as 30 matings which resulted in pregnancy when the dogs were mated only once, are available for study.

We are not as yet certain whether the termination of pregnancy is due to a hormone secreted by the mother or to the natural coming of age of the fetuses. This is not a question which can be settled here; but it does have a bearing upon the determination of the period of gestation.

The age at which puppies open their eyes after birth is one indication which may be used in determining whether a litter is premature or late in arriving, but not a reliable one in all cases. In most instances puppies born from whelpings which occurred as soon as 59 days after coitus did not have their eyes open on the tenth day after birth. Most frequently, in fact, 12 to 14 days passed before the eyes opened.

Gestation may be defined as the period from the moment of the fertilization of the ovum by the sperm to the moment of birth. This is regarded by some as the equivalent of the time elapsing from coitus until the moment of birth. This, however, is inexact, because it is known that ova may live for some time and also that the sperm are able to exist for several days in the reproductive tract of the female, so that one or more days may elapse between the time of coitus and the moment of fertilization.

Most of the tables which have been compiled for the use of dog breeders in calculating the expected time for the arrival of a litter, consider the period of gestation as 63 days. Dukes,[1] however, states that the period is 60 days.

According to Whitney[2] the time of ovulation in the average bitch appears to be well along in the acceptance period. According to Evans and Cole[3] it is generally the first day. A larger series of data, to be presented in a subsequent paper, throws more light on this question. For the purposes of this paper, we may assume that ovulation transpires most frequently about the fifth day of the acceptance period. It would seem, therefore, that the most useful method of determining the duration of the gestation period would be to consider carefully all of the matings which took place at, near, or after the time of ovulation.

As the author showed[2] the sperm have already reached the ovarian bursa by the time copulation is completed. After ovulation has occurred only a short time elapses until fertilization has been accomplished. The ova must then traverse the short Fallopian tubes, move down one to eight inches of each horn of the uterus, become implanted, and develop. That is, of course, assuming that the sperm do not meet the ova in the oviducts or uterus, if, in fact, fertilization may occur in the uterus.

If copulation has taken place before the time of ovulation the sperm must live in the female reproductive tract until ovula-

[1] Dukes, H. H., 1937. The Physiology of Domestic Animals. Comstock Pub'l Co.
[2] Whitney, L. F., 1927. The mating cycle of the dog. *Chase Magazine*, July.
[3] Evans, H. M., and H. H. Cole, 1927. The oestrus cycle of the dog I. and II., *Jour. Anat*, Nos. 10, 11.

tion has occurred. Under such circumstances one would expect that the gestation period would be considerably longer than gestation periods which result from matings which took place the day of ovulation, or after that day.

The records show 13 cases where coitus permitted before the fifth day of the acceptance period resulted in conceptions. The number of days elapsing from a single copulation to parturition are as follows: 63, 62, 62, 62, 65, 65, 64, 63, 64, 64, 67, 66. The average length of these gestation periods was 64.0 days.

Seventeen cases are found when coitus was permitted on or after the fifth day of the acceptance period. These gestation periods are as follows: 62, 62, 61, 62, 59, 59, 59, 63, 61, 62, 63, 59, 59, 59, 61, 60, 61, making an average of 60.7.

Besides these 30 fertile matings, there were 17 infertile ones. In analyzing these 17 it is especially noteworthy that 13 of them occurred on the first day of acceptance.

We do not know whether the day of birth is determined by the age of the puppies, by the bitch, or by a combination of the two. Whichever it is, apparently the gestation period in the bitch can be said to be approximately 61 days and not 63, as many dog breeders have held; on the other hand, it is apparently slightly longer than Dukes' estimate. We can partially explain the 64.0 day average of the 13 cases where coitus occurred before the fifth day by assuming that ovulation had not transpired and that the sperm had to live several days in the reproductive tract of the female.

There is still another bit of evidence which may be useful in helping to answer this question. In a later paper the author will present case histories where one bitch was bred to more than two dogs in order to attempt to determine the time of ovulation. For instance, a bitch was bred to one dog on June 18 and to a genetically dissimilar dog on June 22. The puppies were whelped on August 20th, and sired by both studs (five by one, and six by the other). It is reasonable to assume that the sperm did not live for four days from the time of the first service to the time of the second; but they could have lived for two days, so that some of them could have fertilized the ova, the remaining ova living for two days longer until the second mating. From the time of the first mating to the day of parturition 63 days passed; from the second mating to parturition, 59. In fact, in the case of all these bitches the gestation period was less than 63 days and in the majority of cases the conception took place as a result of later matings.

Summary

From a practical point of view it may be said, on the basis of the author's data, that the gestation period in the bitch is close to 61 days, with some variations. This is computed on the basis that the gestation period is that period of time elapsing between the fertilization of the ovum and parturition. If the gestation period is considered to be the time elapsing between coitus and parturition, then the gestation period in the bitch is about 61 days, when the bitch is bred coincident with or after ovulation. If the bitch is bred before ovulation, then the gestation period is 61 days plus the number of days which the sperm must live until ovulation occurs. Of course, allowance must be made for natural variation in individual bitches.

Intestinal Obstruction in Dogs

That many cases of gastrointestinal obstruction occur as a direct result of feeding entirely inappropriate food to dogs and cats, is an established and indisputable fact. It will be noted that the symptomology remains fairly constant, and a striking basic similarity exists in the four cases reported here.

Case No. 1.—July 7, 1938, after having suffered for several days from anorexia, constipation, intermittent emesis, and flatulence, with acute distention and rigidity of the abdomen, a male Newfoundland dog, aged four years, was hospitalized for the treatment of clinically obvious intestinal obstruction. The animal was very reluctant to move about; the temperature was 102.4°F.; the abdomen was rigid, but palpation revealed an extensive nodular impaction; there was abdominal pain on the application of pressure, and a mild degree of toxemia was noted. Dyspnea was quite noticeable; probably due to diaphragmatic pressure.

The intestines were irrigated with 40 quarts of cold water, and many small bones (ribs and spinal segments), sufficient to fill a pint jar, were obtained. The same treatment applied the following day was productive of more such bones. Two ounces of mineral oil were administered hourly throughout the day until one pint had been given, which was followed by giving one ounce of a mixture of equal parts of a saturated solution of sulphate and elixir digestive compound,* repeated until purging occurred. The patient was discharged July 9, with instructions to feed only a liquid diet of milk and broth for three days. This patient was reëxamined July 18, and found normal.

Case No. 2.—A female Scotty, two years of age, owned by a roadside restaurant operator, was admitted September 4, 1938, presenting a similar clinical complex. Similar treatment was administered, except that a smaller quantity of water was used for the intestinal irrigation, and no mineral oil was given. Many circular pieces of cellophane of various lengths and widths were obtained. The patient was discharged the following day.

Case No. 3.—The patient was a female Airedale, six years of age, admitted October 15, 1938, suffering with a symptom complex similar to the previous two cases. This dog, however, appeared to be suffering continuous severe pain which was not present in either of the foregoing cases or in the case described in the following. Intestinal irrigation and catharsis produced two pounds (weighed wet) of whole grains of yellow corn. This case was discharged October 16.

Case No. 4.—November 18, 1938, a male Boston terrier, ten months of age, presenting similar symptoms, was hospitalized. On following the same treatment routine, the obstruction in this case was found to consist of garden soil and dry leaves. Complete recovery followed, and this patient was discharged November 20, the same instructions being given the owner.

In none of these cases did the temperature reach or exceed 103°F; the degree of lassitude varied from very slight to near-coma, depending on the length of time the obstruction was in existence and the degree of toxemia established. Objective or induced pain was not constant, although pain of some nature or degree could be detected in each animal. Nevertheless, pain does not appear to be necessarily a component part of the syndrome; at least not clinically. Doubtless, pain is in evidence during the early stages of stasis and before the muscles of the intestinal walls become atonic due to fatigue induced by the pressure of flatus and the static intestinal contents. Each case has usually developed to this stage before being presented for treatment. The first case, departing from the routine treatment, was given mineral oil to serve as a protective to the intestinal mucosa and prevent further irritation and entrance of infection to the blood stream through minute wounds undoubtedly left by the bones. The cellophane mentioned in Case No. 2 had been deliberately fed to the dog by the owner, as he stated that much meat adhered to it when removed from bologna, etc., and he did not wish to waste it. This type of cellophane being insoluble in 10% HCL in 24 hours, and only partly soluble in concentrated C. P. HCL in the same length of time, its indigestibility makes it a rather dangerous substance to feed dogs. The dog referred to in Case No. 3 came from a poultry farm, and had had access to grain soiled with chicken blood during the dressing process. Pica, due to mineral deficiency, appears to have been

*This prescription was incorrectly given as "digitalis compound" in Doctor Pritchett's previous article on this subject (*Vet. Med.* 34:11, p. 669).

the aggravating cause in Case No. 4, as this dog was known to frequent a spot in a neighbor's yard where dish-water was discarded, licking and eating the dried chunks of soil.

H. D. PRITCHETT.

Doylestown, Pa.

Injury to the Foot in a Greyhound

The patient, a three-year-old greyhound, had cut its foot on a broken bottle some six months previously, the wound being on the dorsal part of the foot over the region of the metatarsal bones. There was at the time of examination a discharging sinus over the metatarso-phalangeal joint, the discharge having a fetid odor. The radiographs show the condition at this time.

The animal was prepared for operation, and a preliminary dose of ½gr morphine sulphate was given two hours before injecting 8cc of nembutal into the saphena vein of the good leg.

The whole of the remaining portions of the metatarsals, together with the corresponding phalanges, were removed with little difficulty. There was one large sequestrum lying quite free in a mass of pus.

The wound was treated with a standardized Dakin's solution for five weeks, gauze being continually soaked in the solution and plugged into the wound. Healthy granulations soon appeared, and the wound finally closed with the application of a saline solution and a scarlet red ointment.

The animal is not a great deal inconvenienced by the loss of half a foot, and is now, 12 months after operation, capable of chasing rabbits and hares, the purpose for which it is used.

The case is interesting because of the rarity of such extensive osteomyelitic changes in the foot of a dog, and of the rapid healing which took place after the sequestrum was removed.

W. M. NIELSEN

Cessnock, N. S. W., Australia

Diseases of Dogs in the American Tropics

Canine distemper is quite prevalent in the Dominican Republic.

Rabies occurs here, although not every dog that bites suffers from rabies. Vaccination is employed to a considerable extent.

Sarcoptic mange has been encountered more or less frequently.

Various internal parasites are present; hookworm seems to cause the most trouble. No doubt the heartworm exists here, but we have not confirmed its presence as yet.

Ear canker is seen rather frequently in both mild and severe, stubborn cases. The latter used to worry us greatly, but we hope for better results in the future. I spent six weeks applying local remedies, without benefit, in a case in a Spitz, which the owner said my predecessor had treated for over a year. Then I obtained drainage by removing a v-shaped piece of skin at the lower commissure of the ear. Two weeks later the wound had healed, and there has been no return of the trouble.

GUY A. ROBERTS

Ciudad Trujillo, D. R.

Wildcat Lacerations in a Chow

July 31, 1939, a red male chow, four years of age, was brought to the Stewart Veterinary Hospital at Colorado Springs, Colo., for treatment, with the following history: July 4, 1939, the animal had been clawed and bitten severely about the head and face by a wildcat. It had received some treatment from another veterinarian.

Examination revealed a corneal ulcer of the right eye resulting from a wound on the cornea at the time of the accident. A copious purulent discharge was draining into both eyes from the region of the medial canthus of both orbits. A severe conjunctivitis was evident in both eyes. General depression, shagginess of coat, anorexia, and a lack of appetite were noticed. It was possible to pass a probe from one eye to the other through the fistulous tract under the skin and connective tissue, and to expose the frontal bone with the probe.

The patient was placed on the operating table, and the operative area between the eyes was shaved, cleaned, and disinfected with 70% alcoholic solution. A 1% procaine solution was infiltrated into the area, completely anesthetizing it. An incision three-quarters of an inch long was made and drainage was established. However, when the wound closed five days later, the purulent discharge began again from both eyes.

The patient was again placed upon the operating table and the same preoperative measures were taken. An incision was made and a section of skin and subcutaneous tissue three-eighths of an inch wide and one inch long was removed. Necrotic tissue was removed laterally both ways from the incision. The section of the frontal bone which could be exposed was curretted and the wound was irrigated with aqueous solution of zephiran and packed with gauze. The next day it was again irrigated, with the same solution and packed with an antiseptic jelly containing 0.4% allantoin. This treatment was repeated daily for five days. An eye ointment containing 5% argyrol was used in both eyes and the skin was greased with petrolated jelly around the wound and below the eyes to relieve the skin irritation from the discharge.

The patient was released from the hospital two weeks after entering. The general appearance and attitude were excellent, and the eyes were clear, except for a small spot on the cornea which has since cleared. The incision had closed nicely and discharge had stopped completely.

The chow, which was of a mean disposition, was at first handled at the hospital with some difficulty, but after his confidence had been won, he was handled with ease. Much attention was given to the treatment of the fistulous tract, to prevent damaging any more of the optical tissue; for this reason only mild antiseptics were used.

PAUL T. LOYD, K.S.C. '40.

Manhattan, Kans.

* * * *

Ringworm and other fungus infections in dogs respond nicely to 6% chlorine rinses. —*C. L. McGinnis.*

* * * *

Ear mites in dogs are easy to diagnose. The acari may spread from the ears to all parts of the body but more commonly to the face than elsewhere. Canex is the remedy of choice. Drop it, full strength, into the ear every other day. When the mites in the ears are killed they soon disappear from the remainder of the body. An application of vaseline will hasten their disappearance, or derris powder will make short work of them.—*C. L. McGinnis.*

* * * *

Nephritis is a very common finding in our dog post-mortem cases—about 10% of all cases. They fall roughly into the two main groups, chronic and acute, and eventually the dogs are destroyed suffering from uremia. The acute cases may be very sudden in onset and are sometimes associated with gastritis or gastroenteritis, and buccal necrosis of the "Stuttgart" type and in a few cases with necrotic endocarditis. We have not been successful in isolating spirochetes from any of these cases so far.—*Institute of Animal Pathology* (London).

Book Reviews

The Practice of Veterinary Medicine. *By D. H. Udall,* Professor of Veterinary Medicine and Superintendent of the Ambulatory Clinic, New York State University. Third edition, completely revised. Cloth bound. 672 pages; 102 illustrations. Published by the author, Ithaca, N. Y., 1939. $6.00.

"Udall's Practice" has made such a definite place for itself with practitioners and students of veterinary medicine that further reviews of the frequently appearing revisions are supererogatory. A simple announcement that a new edition is available suffices. However, it is desirable to state that the type for this new edition has been entirely reset, the text considerably extended, and improved. The change in the typography and format, which are now of the conventional text book style, is a welcome improvement with this edition.

One salutary effect of the revision is omission of the preposterous statement of earlier editions that the increase in rabies in this country is probably due to the increased use of anti-rabies vaccine. The discussion of rabies is still open to criticism in that it digresses to interpose an array of evidence that the one-dose prophylactic vaccine is a failure, omitting a greater quantity of evidence that it is highly efficient, and even failing to mention that such evidence exists. The controversy over anti-rabies vaccine may be dismissed with the general statement that those who have used it in the field laud it and those who have tested it in the laboratory damn it. For the present, writers, unless they discuss it from personal experience, will spare the patience of readers by so limiting themselves. The explanation for this conflict lies in the future. It is idle to attempt it with the information now available.

The demand for "Udall's Practice" which has made possible its frequent revision is a most encouraging sign. A decade ago the attitude of the profession was most discouraging to prospective veterinary authors. Udall's experience shows that if a new veterinary book "clicks," there will be a healthy demand for it.

The Diseases of the Genital Organs of Domestic Animals. *By W. L. Williams,* Professor Emeritus, Cornell University. Second Edition. Cloth bound. 617 pages; 199 illustrations, 3 in color. Published by the author, 209 White Park Road, Ithaca, N. Y., 1939. $9.00.

Although the reviewer has enjoyed immensely reading this work, he must confess being at a loss as to how to go about reviewing it. There is so much in it that differs from and goes beyond ordinary veterinary thought and beliefs in the matter that comparison fails and the limitations of space preclude a detailed enumeration of new features and new viewpoints. Veterinarians engaged exclusively in small animal practice and in food inspection will find it of only academic interest, but those in general practice, in disease control from a regulatory standpoint, in research and in teaching—all interested in the physiology and pathology of the reproductive processes of farm animals, will find it a thought stimulator.

The author appears to have launched upon the preparation of this work in a spirit of adventure. He has recorded his observations, keenly and discerningly made, with entire freedom and given some of his conclusions based thereon with just as little inhibition by the dicta of orthodoxy. Possibly the author has expected some hostility to his treatise, for he has included 13 tables of data around which the discussion largely revolves and from which his conclusions that depart from current beliefs are drawn. Thus the critic will have to support contrary opinions by an equal or greater accumulation of data or tacitly admit his opinions are without firmer support than theory.

Thirty years ago the author published "Veterinary Obstetrics, Including the Diseases of Breeding Animals and of the Newborn," which marked an advance over previous publications on this subject in this country and Europe. A score of years later the work was extensively revised and separated into two parts, "Veterinary Obstetrics" and "Diseases of the Genital Organs of Domestic Animals," embodying the new knowledge developed by almost constant

study of the author during this period and by the work of others.

In the present work the subjects are combined in a measure, since "Veterinary Obstetrics, which largely revolves about dystocia, is inseparably bound up with the various pathological phenomena of the reproductive system." Dystocia, even that due to mal-position of the fetus and to monsters, is regarded as as truly a result of genital disease as is abortion, retained placenta, or sterility. The discussion is enriched with the observation and study of the author's 60 years in the veterinary profession and with his entire familiarity with the literature on the subject, both in this country and in Europe.

The subject is approached always from the clinical side. No extended discussion of the bacteriology of genital infection is included. In the author's view, infection is a secondary factor in genital disease, the result of poor hygiene, improper breeding practices, or insanitary methods, or a combination of two or more of these. The remedy lies not in attacking the secondary factor but in correcting the basic fault or faults.

The author is skeptical about the claims for artificial insemination, finding the evidence adduced in its favor far from supporting the claims made for it. He regards the resort to artificial insemination in the case of animals incapable of breeding because of disease (*e.g.*, arthritis) as being a questionable practice; and in the case of physical deformity or advanced age as undesirable, since quite enough monstrosities and weaklings are produced by natural mating. In any case, "the value of pregnancy is grossly overemphasized"; it may be in fact a liability. Vigorous offspring is the desideratum. "It is highly essential to dissociate conception and economic reproduction."

The scope of the work is indicated by the chapter titles, which are: Hybridization; Inbreeding; Teratological and Pathological Ovulation and Spermatogenesis; The Influence of the Health of the Newborn upon its Fertility as an Adult; The Breeding Season; The Age at which Young Animals Should First be Bred; The Breeding of Old Animals; The Rapidity of Breeding; Foods and Feeding; Labor, Housing and Confinement; Estrum; The Artificial Induction of Heat; Copulation, Coitus; Artificial Insemination; Pregnancy; Malignant Tumors; Benign Tumors; Animal Plagues; Genital Tuberculosis; Specific Infections of the Reproductive System; Bacterial Invaders; Molds Associated with Interferences with Reproduction; Protozoan Diseases Interfering with Reproduction; Virus Diseases of the Reproductive System; Unclassified Infections of the Reproductive System; The Diseases of the Male Reproductive Organs; The Diseases of the Female Reproductive Organs; Chronic Diseases of the Uterus, Cervix, Vagina and Vulva of the Non-Pregnant Female; Displacements of the Gravid Uterus and Vagina; The Diseases of the Gravid Uterus and of the Contained Ovum; The Pathological Phenomena Surrounding the Birth Period; Parturient Injuries; Puerperal Displacements of the Urino-Genital Organs; Puerperal Diseases; Asphyxiation of the Newborn; Sepsis of the Newborn; Antenatal and Early Postnatal Ruptures of Tendons and Ligaments; The Impaction of the Large Intestines with Meconium; The Physiology, Pathology and Hygiene of the Digestive System of the Newborn.

The 13 tables referred to in the foregoing are: I. Breeding Record of 18 Heifers Bred to Calve First in 1912; II. Breeding Records of a Diseased, Compared with Healthy Bull, in a Purebred Herd; III. Breeding Records in a Stud During 1920-21; IV. Breeding Records of Three Hereford Bulls During Ten Years; V. Breeding Behavior of Two Jersey Bulls in a Herd; VI. The Duration of Gestation and the Mortality of Bovine Twins According to Sex; VII. The Breeding Efficiency of Mares According to the Duration of Time They Were Carried in Utero as Fetuses; VIII. Breeding Record of Female Progeny of Bull X (Wagner); IX. Changes in the Fertility of Heifers Following Improvements in the Feeding as Young Calves; X. Breeding Records of a Holstein-Friesian Herd from 1920 to 1933 Inclusive, Showing the Influence of Rainfall upon Fertility; XI. The Duration of the Estrual Cycles in a

Group of 24 Consecutive Heifers from a Bang-Negative Purebred Dairy Herd; XII. Breeding Records of Nine Cows Showing the Effects of Rapid, Compared with Conservative Breeding. XIII. The Persistence of the Positive Reaction to the Agglutination Test for *Bacillus abortus*, from Calfhood to Breeding Age.

* * * *

Atlas and Dissection Guide For the Study of the Anatomy of Domestic Animals. *By H. L. Foust.* Paper bound. 64 pages, illustrated. Collegiate Press, Ames, Iowa, 1939. $1.75.

This is a handy-size atlas consisting of a group of plates that the author has found useful in presenting the subject of veterinary anatomy to veterinary students; an effort being made to place the responsibility for learning upon the student.

It contains 28 full page plates showing dissections; 16 of the horse (five of them in two colors), six of the ox (two in two colors), one of the pig (in two colors), two of the dog (one in two colors), two of the chicken (in two colors), and one plate showing the comparative structure of the horse, cow, pig, and dog. The various structures shown in the dissections, usually about 100, are numbered, and opposite each plate a key to the numbers is given.

The dissections and the art work are excellent. The atlas will prove highly useful to practitioners as well as to students.

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Microscopic Diagnosis of Parasitism in Domestic Animals. Laboratory of Animal Pathology and Hygiene, University of Illinois. Paper bound. 124 pages; profusely illustrated. Circular 496, Agricultural Experiment Station, College of Agriculture, University of Illinois. Urbana, Ill., 1939. $1.00.

This bulletin is a revision and extension of one of the same title published several years ago by the Illinois station (Cir. 401). In this bulletin more parasites are described and many additional illustrations included. The treatise is quite comprehensive, containing directions for procuring specimens for examination (feces, skin scrapings, mucus, urine), their preparation, selection of equipment for the examination and the modus operandi of the examination. All steps in the collection, preparation and examination of specimens are illustrated, as are also the adult parasites and the eggs. Halftone illustrations are shown of a considerable number of parasitized organs or tissues and of animals suffering from parasitisms. A new feature of this bulletin is a schematic representation of the life cycle of the parasite, with suggestions for control measures. The bulletin includes parasites of horses, cattle, sheep, swine, poultry and dogs. It will prove a useful manual for the practitioner or other diagnostician in the matter of animal parasitisms.

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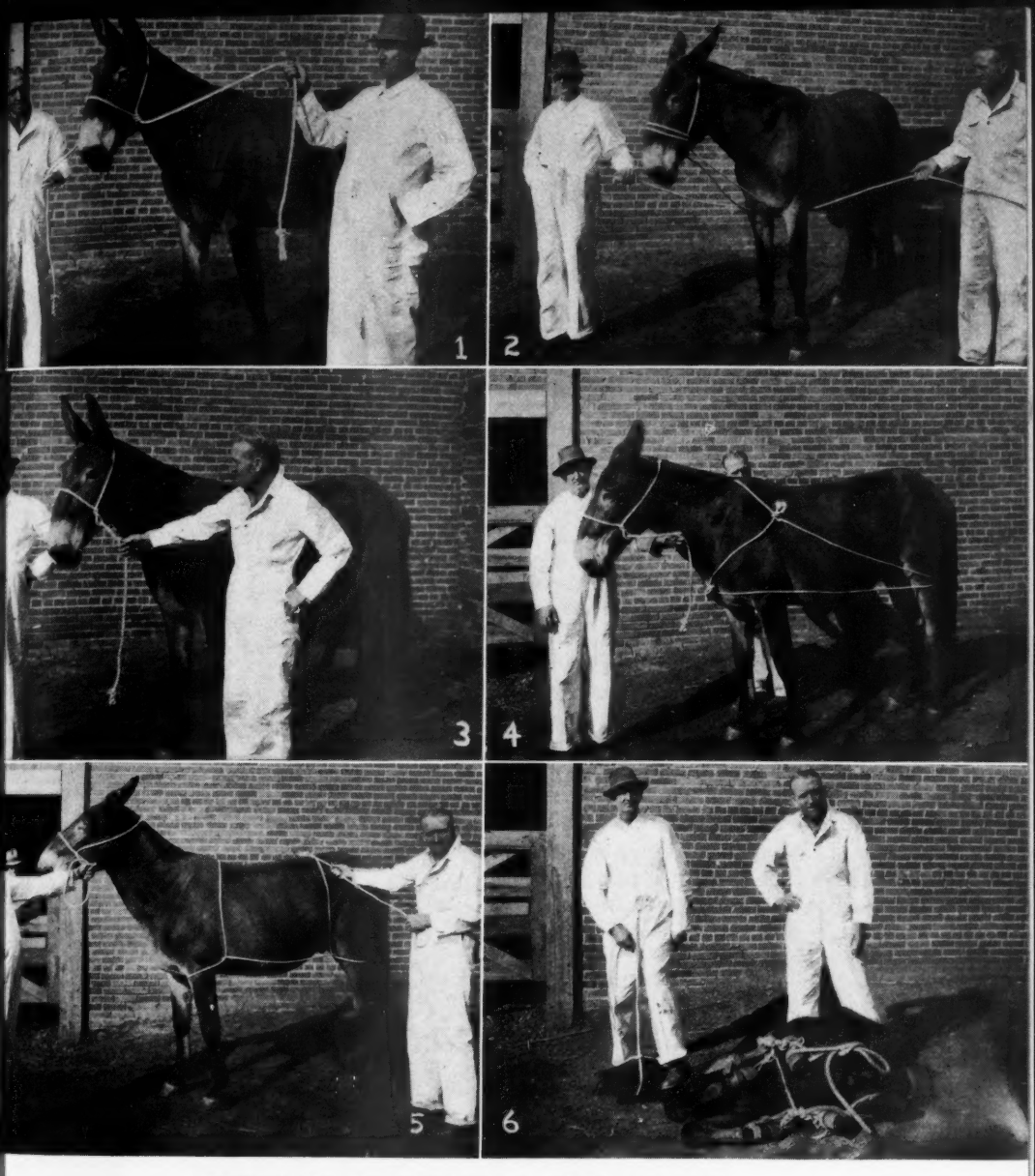
Laws About Dogs. *By Capt. Will Judy,* Editor of Dog World Magazine. Paper bound. 16 pages. Third Edition. Judy Publishing Company, Chicago, 1939. 25c.

This is a most useful little manual. Exceedingly brief, but its brevity is one of its merits. Information sufficient for the layman is to be found readily on almost any phase of the broad subject discussed. The author is a lawyer and with his intimate knowledge of dogs and the dog industry is peculiarly fitted to discuss "laws about dogs." The information this pamphlet contains gives the practicing veterinarian an insight into certain of his own rights and particularly his liabilities in the treatment, boarding, care and killing of the animals of his clients and of those brought to him in emergencies by other than the owners. The scope of the work is indicated by the following subdivisions into which the discussion is classified: History and Sources of Canine Law; Title of Ownership in a Dog; Licenses and Taxes; Justifiable Killing of Dogs; Restraint, Quarantine, Examination, Inoculation; Protection of Rights of Dog Owners; Liability of Dog Owner for Acts of His Dog; Laws against Cruelty; Laws Particularly Affecting Breeders, Kennelmen and Exhibitors.

"Laws About Dogs" contains the answers to many of the questions asked veterinarians, or at least can be referred to in lieu of an answer.



METHODS FOR THE RESTRAINT OF HORSES AND MULES

Fig. 1. A dental halter. By a rope on each side, the head can be cross tied, immobilizing it.

Fig. 2. A "bronc" halter, for use with animals that have a tendency to rear. By placing the ropes under the front legs in this manner it is possible to make the animal turn a somersault with very little effort; after this occurs once, it loses its desire to fight. Pulling the ropes brings pressure on the jaws, in a way that is more effective in some cases than a twitch.

Fig. 3. A draw halter with no knots. This will fit a dog, hog, or mule—the size doesn't matter.

Fig. 4. For a kicker. The restraint is an improvement upon the side line.

Fig. 5. Hippo-harness made with a lariat. This keeps the animal from pawing, striking or kicking. It is a good harness for breeding and obstetrical work, for dentistry on obstreperous patients, for dressing sore necks, for mounting horses that resent being ridden, and many other purposes. Useful for cattle also.

Fig. 6. Casting harness, on a four-month-old colt weighing 274 pounds. This harness was used at the Memphis clinic of the A.V.M.A. last summer.—*W. L. Stroup, Corinth, Miss.*

The Veterinarian in Cyprus

Cyprus, a British colony, is a roughly parallelogrammic island with an area of 3,584 square miles, located in the eastern Mediterranean. The population is slightly over 310,000.

The government veterinary service is one of the most efficient in the British empire, and there is only one veterinarian engaged in private practice.

The veterinary service is responsible for the control of contagious disease, the inspection of live stock prior to export, the quarantine control of imported animals, the inspection of dairies, the supervision of meat inspection in the principal towns, the registration of stallions under the Horse Breeding Law, and the inspection of and veterinary attendance on police horses and government-owned live stock. Advice is also given to private stock owners with regard to the housing, feeding, breeding, and general management of their animals. The members of the field staff also give advice and assistance in cases of non-contagious disease and accidental injuries.

Drugs and vaccines required for the control of contagious and parasitic diseases are supplied free of charge, as the majority of the stock owners are peasant proprietors whose animals represent virtually all their capital.

The general condition of all kinds of live stock in Cyprus depends largely on climatic conditions. Mild weather in January and February following a satisfactory rainfall in the previous months produces a plentiful supply of natural pasture, and the milk supply in sheep and goats is above average. High humidity in August, however, heavy rains in mid-September, with no further rainfall until the end of November, then very heavy rain and exceptionally cold weather to the end of the year, complete a sequence of the factors which are responsible for severe mortality in sheep and goats. Under such conditions, parasitic gastroenteritis and lungworm disease take a heavy toll.

To overcome these difficulties, large amounts of copper sulphate and nicotine sulphate are used, and an additional veterinary staff is employed to distribute drugs free of charge and demonstrate the methods of dosing. No drugs, however, are of much value in the absence of sufficient nutritive feed.

Aside from parasitic infestations, the incidence of specific diseases is small in Cyprus. Now and again there is a case of anthrax, but the annual vaccination of sheep and goats against this disease has yielded very satisfactory results in the Island.

The government veterinary hospital at Nicosia, the capital, has accommodations for twelve large animals and a varying number of smaller stock, depending on the number of experimental animals under observation.

There are 200 police horses in the Island, each member of the mounted branch providing his own mount, for which a veterinary certificate of soundness and suitability is required before it is enrolled. The horses as a whole are well cared for; the majority of the ailments are due to overfeeding and lack of exercise during periods when the animals are not on patrol. Horses are castrated by the emasculator method under chloroform anesthesia; bulls, rams, billy-goats and burros, the principal beasts of burden, by the Burdizzo method.

The conditions under which poultry are kept in the villages in Cyprus are such that heavy losses frequently occur from outbreaks of fowl cholera, fowl typhoid, fowl pox, and spirochetosis. As a rule the poultry are allowed to wander around the streets and fields by day, and at night are closed up in small, insanitary sheds which are almost impossible to keep clean and tick-free. Dead birds are frequently thrown out into the streets or onto waste ground to which other poultry have access. Much education in the general principles of poultry sanitation is required before the diseases of poultry in the Colony can be brought under control.—*Walter Buchler.*